ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL

A THESIS SUBMITTED TO BABA FARID UNIVERSITY OF

HEALTH SCIENCES, FARIDKOT (PUNJAB)

IN THE PARTIAL FULFILLMENT

FOR THE AWARD OF DEGREE OF

M.Ch

IN

PLASTIC & RECONSTRUCTIVE SURGERY

MAY, 2023

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Dedicated

to

my Family

&

Teachers

ACKNOWLEDGEMENT

As I sit down today to pen the conclusion of my thesis, I think of the memories of this beautiful journey of M.Ch and all the people, without whose support this would not have been possible.

First and foremost, I am grateful to God for providing me this opportunity and giving me courage and strength through the ups and downs of this journey.

I would like to extend my deepest gratitude to my Guru, my guide and my supervisor, ***Dr. Rajinder Mittal*** *Sir (Professor and Head, Department of Plastic & Reconstructive Surgery, DMC&H, Ludhiana) who made this work possible. His guidance and advice carried me through all the stages of writing my thesis. His expertise in the subject, particularly microsurgery, has made this an incredible learning experience. I would like to thank him for sparking my curiosities, and being a tremendously kind mentor who has made my residency a truly enjoyable experience for me. Thank you, sir, for your constant support and trust in me*

The completion of this thesis would not have been possible without the encouragement of my co-supervisor, ***Dr. Sheerin Shah*** *mam (Associate Professor, Department of Plastic & Reconstructive Surgery, DMC&H, Ludhiana). With great regard, I acknowledge her guidance and support throughout this*

process. Her interest in our project, attention to detail and endless patience helped give shape to my thesis. She has taught me sincerity and hard work go hand in hand with a happy personal life. Thank you, mam. You are a great motivation for many.

I would also like to extend my respect and gratitude to ***Dr. Ramneesh Garg*** *sir (Professor, Department of Plastic & Reconstructive Surgery, DMC&H, Ludhiana). Sir helped me identifying my own strengths and weaknesses. The opportunities he gave me, boosted my self confidence in surgeries. My sincere thanks.*

A big thanks to ***Dr. Sumit Gupta*** *sir (Consultant, Head and Neck Oncosurgery, DMC&H, Ludhiana) for providing opportunities and unconditional support to complete my thesis.*

I would like to extend the warmest thanks to my exemplary mentor, ***Dr. Dhirendra Suman*** *sir (Professor and Head, Department of Burns and Plastic, UCMS, Delhi) and* ***Dr. Govind*** *sir. I am forever indebted to them for their immense contribution to my academic journey. It's been a privilege to have learned under their guidance. Had I not met them, I would not have come so far in this field.*

This endeavour would not have been possible without the guidance of my seniors ***Dr. Karan Singh*** *and* ***Dr. Devika*** *and*

the support of my colleagues and my friends ***Dr. Sumit Singh Gautam, Dr. Bhavuk , Dr. Ojassvi Rishi, Dr. Deepika Sharma, Dr. Anupreet, Dr. Kayenaat*** *and* ***Dr. Poornima.***

To the love of my life ***Dr. Navdeep Kumar Singla*** *(Navvi) and our baby* ***Nithwik Singla*** *(my Moushie), without your support it was impossible. Hats off to the sacrifices you both made. Love you both. Thank you, Navvi, for helping me survive all the stress and always knowing how to make me laugh. To my family, thank you for always loving and supporting me even at my worst. To my parents,* ***Dr. N. K. Singla, Mr. Naresh Kumar Verma, Mrs. Sunanda Singla*** *and* ***Mrs. Neelam Verma****, thank you for your unconditional love and strength through the toughest phases of my life. I feel truly blessed to have parents who have given me these invaluable opportunities to be happy and successful in life. Thank you for shaping me into the person I am, and I hope it is someone you can be proud of. To my siblings,* ***Ms. Rishu*** *and* ***Mr. Varinder Madhav****, thank you for always tolerating me. You have been a constant source of encouragement, warmth and love. Thank you for your constant, sometimes unsolicited, life advice and words of wisdom. A special thanks to my sister-in-law* ***Mrs.Parul*** *(Shavi Di) and brother-in-law* ***Mr. Mohit*** *(Jiju) and my Musketeers* ***Yuvaan*** *and* ***Shivaye*** *for their immense love and support.*

This important milestone in my life would be incomplete without mentioning my friends. ***Dr. Kiranjot Kaur*** *and* ***Dr. Manmeet Singh****, thank you for being my constants throughout this journey.*

I bow my head in sincerely acknowledging all my patients who consented to become a part of this study. Without their cooperation, this thesis would not have seen the light of day.

A special thanks to ***Mr. Harish, Mr. Sarju*** *at my Department for their help and support and also to* ***Mr. Naveen, Mrs. Namita Bansal*** *and* ***Mr. Gurinder Singh*** *at Research & Development Centre, DMC&H, Ludhiana for their sincere efforts in a fine reproduction of this work.*

LIST OF ABBREVIATIONS

ALT	Antero lateral Thigh
BK	Below knee
Hr(s)	Hour(s)
L/G	Lactate/Glucose
LGR	Lactate/Glucose Ratio
LD	Latissimus Dorsi
MD	Microdialysis
RSA	Reverse Sural Artery
SAS	Same as surroundings
TFL	Tensor Fascia Lata
WNL	Within Normal Limit

ABSTRACT

Introduction: The visible improvement in the success rate of flaps is attributed to several reliable flaps monitoring tools. Clinical assessment is still the gold standard but it utilizes subjective parameters. Several objective methods have thus evolved over time. Amongst them, capillary lactate and glucose measurements from flap for predicting flap survival and need for re-exploration is gaining importance due to its objectiveness, simplicity, non-invasiveness, efficacy and cost effectiveness.

Aim of Study: To study the role of capillary Lactate/Glucose ratio as a predictor of Flap Survival.

Materials & Methods: A Prospective study was conducted on all patients fulfilling the inclusion criteria, who underwent any kind of flap surgery. After the surgery, along with clinical monitoring, flap Lactate and Glucose was recorded at 0, 1,3,6,12,24 and 48 hrs. Values were used to calculate LGR. Two groups were made- Group A: Flaps with no post-operative complications and Group B: Flap with post-operative complications (signs of necrosis, need of any surgical intervention)

The L/G ratio (LGR) of 2 groups was compared and data was utilized to calculate the cut off value of LGR at which flaps begin to show signs of non-viability.

Results: Over a period of 17 months, 63 flaps were included. With 12 flaps in Group B, the cut off value for lactate was>= 6.4 mmol/L, for glucose cut off attained was <=6.2mmol/L and for LGR the cut off of 0.98 was attained. LGR was found to be better predictor than individual glucose and lactate and also critical cut off was reached well before the appearance of clinical signs. The mean cost for 48 hours of monitoring was less than INR 2500. We conclude that LGR can be used an a sensitive, objective, efficient and cost-effective tool for predictor of flap viability and can aid salvage dying flaps.

Keywords: Flap Monitoring, Objective, Lactate/Glucose ratio, cost effective

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INTRODUCTION

Flaps are the workhorse of Reconstructive and microvascular surgery. A flap is a unit of tissue which is transferred from a donor to a recipient site with its blood supply.[1] Optimal blood supply is essential for flaps to survive, and compromised supply, if detected early, can help salvage a dying flap.[2]

Minimizing flap failure is a key goal for any Reconstructive and Microsurgical unit.

It is well known that congestion due to venous thrombosis is more common and congested flaps are more prone to fail than arterially compromised flaps.[3-8]

Data suggest that the commonest cause of free flap failure is a problem with the venous anastomosis, occurring within the first 24 to 48 hours post operatively.[9,10]

The success of a free flap salvage operation is dependent on the early detection of vascular compromise and the subsequent short take-back time to the operating room.[11]

In case of pedicled flaps, the causes may be hematoma, external compression by tissue odema or tight Dressing, twisting of pedicle, and thrombosis leading to venous congestion which if prolonged can lead to flap necrosis.

The visible improvement in the success rate of flaps is attributed to the advancement of microsurgical techniques, instruments and invention of several reliable flaps monitoring tools. Thus, flap monitoring helps early detection of compromised flap which further aids in flap salvage.[12] An ideal monitoring method should be harmless to the patient and the flap, accurate and reliable, capable of providing rapid response to circulatory change, capable of being used by all personnel, reproducible, inexpensive, and portable.[13]

There are several methods of flap monitoring that have been described. Clinical assessment is still the gold standard which utilizes

parameters such as temperature, colour, turgor and capillary refill. Temperature and colour of flap similar to surrounding skin, capillary refill time less than 3 seconds indicate healthy flap. A pale coloured, cold flap with increased capillary time indicates arterial insufficiency whereas, a warm dusky coloured flap with decreased capillary refill time may be suggestive of venous compromise. However, In dark skinned individuals skin and capillary refill may be masked by deep pigmentation. Another method is the assessment of colour of blood that oozes from the flap on pin prick. Bright pink ooze represents a healthy flap, whereas dark purplish ooze reflects compromised venous perfusion of flap. No bleed on pin prick may be suggestive of arterial insufficiency. These are all the subjective parameters and may be faulty in case of inexperience or factors like inadequate lighting or poor visibility. Also, the results of a literature prove that clinical evaluation alone is unfavourable for postoperative flap monitoring as clinical signs take time to become evident.[3]

Patients who had undergone particularly head and neck reconstruction often have a free flap inside the oral cavity, making clinical assessment of flap difficult.

Several objective methods have thus evolved over time- Doppler ultrasonography of the vascular pedicle[14], measurement of tissue oxygenation[15], analysis by microdialysis[16], use of intra-arterial or intravenous catheters[17], and quantitative spectral imaging (which measures distance, oxyhemoglobin, deoxyhemoglobin, total hemoglobin, and tissue saturation)[18], tissue pH monitoring, skin temperature probes, near infrared spectroscopy, photoplethysmography as well as the use of fluorescent dyes amongst many others.

Amongst different methods, transcutaneous oxygen tension is one of the oldest methods of assessing flap viability.[19,20] However, many other systemic factors affecting oxygen transport and tissue oxygenation can affect the results.

Doppler ultrasonography is a simple method, commonly used for evaluation of blood flow in flaps.[15,21] But it cannot differentiate between the signals coming from adjacent normal vessels deep to the flap.

Laser Doppler study, another method of measuring skin blood flow, can be used for non-invasive, continuous monitoring of skin flaps.[12] Major limitations like sensitivity to position, sensitivity to temperature, shallow penetration and small sample area limits its clinical utility.

Assessment of skin perfusion using fluorescein dye is another method where an intravenous bolus of fluorescein dye is given and tissue is then examined under an ultraviolet lamp.[22,23]

Tissue that is adequately perfused fluoresces under an ultraviolet lamp, whereas inadequately perfused does not. But this method usually underestimates the amount of tissue that will ultimately survive. Also, serial use of this method is also cumbersome.

Continuous pH monitoring of a flap is another method of assessment of metabolic function. Anaerobic metabolism in flap causes rapid fall in pH due to vascular. Compromise.[24] This method successfully used in experimental studies but has never reached widespread clinical application. All these methods require complex apparatus and are expensive.

Mechanism of Microdialysis is based on monitoring the metabolic changes in the flap by detecting changes in glucose, lactate and pyruvate. Tachi et al said tissue glucose level decreases earlier and faster than body glucose level. They hypothesized that when venous congestion occurs, the tissue becomes hypoxic, and anaerobic glycolysis is accelerated, resulting in tissue glucose depletion.[25]

Based on these, several studies have utilized flap glucose and lactate measurements as a flap monitoring tool for predicting flap survival and need for re-exploration. These are more objective, simpler, need no expertise and are cost effective.

Even after successful free flap transfer, there can be periods of transient or permanent ischemia in post operative period which need to be diagnosed at the earliest since prolonged ischaemia may result in flap loss or distal necrosis of flap. Despite so much evolution, the success rate has never reached 100% even in larger series.[4-6] The main reason being pedicle complications.[4,5,12] Studies report that a re-exploration is needed in 6 to 17% of microvascular flaps when perfusion failure is suspected.[26,27]

The literature in the field of microvascular transfer has always been concerned to search for an effective diagnostic tool for pedicle related complications. Amongst them, microdialysis has gained quite attention.[15,16] Simplification has been done by measuring capillary glucose and capillary lactate levels by use of glucometer and lactometer making it a rapid, effective, simple, easy to implement, in-expensive and not needing expertise or big learning curve.

Few studies have reported that the relationship among the two can be superior to individual values.

The aim of our study was to compare the gold standard clinical assessment and intracapillary Lactate to Glucose ratio in predicting flap survival and to assess the superiority of the ratio to the individual values.

AIMS AND OBJECTIVES

To study the role of capillary Lactate/Glucose ratio as a predictor of Flap Survival.

REVIEW OF LITERATURE

Flap monitoring is very significant to a plastic and reconstructive surgeon. The survival of the flap (free as well as pedicled) and the success of the surgeon depends not only upon the surgical technique and the experienced hands but also the post-op flap monitoring and timely decision to re-explore in addition the general condition of the patient.

Though the success rate of free flap is reported to be 95% or higher, the problem of anastomosed artery or vein remains a major cause of tissue loss.[13] And in general, venous compromise or congestion is more common cause of flap failure than arterial thrombosis. Other causes being pedicle complications such as extrinsic compression or kinking.

Most of these complications usually occurs within 48 hours of surgery, thus demands early detection followed by timely intervention before permanent damage sets in. This needs close monitoring of flaps.

The salvage rate of postoperative vascular complications is between 0 and 100%.[6,7] Earlier the salvage surgery is performed, more is the success rate.[8]

Although Clinical Evaluation still remains the gold standard which include Colour, temperature, turgor, Capillary refill time and pin prick to check the colour and character of bleed but this is a subjective observation and requires experience. But it is often too late when these clinical signs become apparent.[28] Various other objective methods are available which are not cost effective.[14-18] Amongst them, the microdialysis which analyses metabolic changes in flap, has shown that Capillary glucose level drops at the time of congestion or ischemia. Within flap blood glucose measurement cannot be relied upon for ischemic flaps, as sufficient blood cannot be obtained to measure the glucose level in a pinprick test. Dynamics of blood glucose concentration in flaps during the postoperative period is such that a single reading of flap's blood glucose is not a reliable indicator of vascular compromise. However, mean of repeated measurements of flap's blood glucose levels taken at different

time intervals can help in deducing the venous compromise.[28] Exact mechanisms responsible are unclear. Sakakibara et al[29], in 2010 hypothesized that in venous thrombosis, flap's blood glucose concentration is reduced due to continual uptake of glucose from the stagnated blood. Venous thrombosis might decrease the inflow of blood from flap's artery by increasing venous and capillary pressures. Lack of adequate blood flow may cause continued uptake of glucose from a slow-moving blood resulting in a local hypoglycemic state. Further, reduced blood flow leads to consequent increase in anaerobic metabolism. The anaerobic pathway uses glucose inefficiently, which further worsens local hypoglycemia.[30]

In 1998, Jonas Rojdmark et al studied the metabolism of myocutaneous flaps using microdialysis and observed a change in metabolic pattern signalling flap ischaemia.[31]

In 2010, Sakakibara et al. [29] reported a lower blood glucose level in congestive flaps in clinical cases.

In 2011, Hara et al [28] described blood glucose measurement for flap monitoring and reported that the blood glucose level of 62mg/dl is the best cutoff value for detecting venous thrombosis, at which the sensitivity and specificity are 88% and 82%, respectively.

In 2015, Bashir et al[32] determine the diagnostic accuracy of blood glucose measurements within flaps in early detection of venous compromise. 127 pedicled and free flaps, including replants, performed on patients between 12 and 60 years of age. Within flap blood glucose measurements were performed using pinprick and a blood glucometer at 0, 6, 12, 24 and 48 hours after operation. Daily examination for clinical signs of venous compromise was used to determine flap viability for up to 7th day after operation. A cut-off value of 62 mg/dL was determined using a receiver operating characteristic curve. Using this cut-off value, the sensitivity and positive predictive values of within flap blood glucose for determining venous compromise were 90% and 91%, respectively,

whereas the specificity and negative predictive values were 78% and 76%, respectively. The overall diagnostic accuracy of within flap blood glucose was 87%.

In 2016, A study done by Muthulakshmi et al[33] on 50 patients who underwent BK amputations, 5 patients developed postoperative flap necrosis due to venous thrombosis. Flap blood glucose level was measured at 0,6,24 hours. Blood glucose levels <62 mg/dl, for detection of flap survival had a sensitivity, specificity and positive predictive value of 86.66%, 100%, 100% at 0 hour, 100%, 100%, 100% at 6 hours and 97.77%, 100%, 100% at 24 hours respectively. The study confirmed a decrease in capillary glucose in all 5 flaps which underwent necrosis and gangrene in the first 24 hours after surgery.

Several reports described the effects of blood flow obstruction on blood glucose and lactate values. In 1989, Heden and Sollevi [34] showed that tissue hypoxia caused lactate values to increase and blood glucose values to decrease at the same time during ischemia.

In 1998, Rojdmark et al [31] measured intra-flap blood glucose and lactate values to evaluate flap blood flow. In 2008, Sorensen [35] used those values for free jejunum flaps.

In 2004, Setala et al [36] reported that blood glucose values decreased 2 to 8 hours before clinical findings emerged, and lactate values increased 1 to 12 hours before the findings of congestion. They speculated that the tissue was in a transient anaerobic metabolic state until the post-reperfusion flap tissue adapted to the new perfusion pattern. Unlike lactate values, after the blood glucose values increased to higher than the upper limit for temporary decreases, they remained higher than the reference value. They also concluded that decreasing glucose levels and increasing lactate concentrations were associated with arterial and venous occlusions from the first hour of ischemia. In venous ischemia, lactate concentrations remained lower than those in arterial ischemia. The

increase in lactate-to-pyruvate and lactate-to-glucose ratios was related to ischemia and also discriminated arterial occlusion from venous occlusion.

In 2006, Setala et al[37] used microdialysis (MD), a method developed to assess in situ tissue metabolism, in the follow-up of 80 consecutive microvascular flaps from October, 2001 to October, 2003. Of the 78 flaps with postoperative data, 58 flaps were uneventful clinically and using MD, and served as the reference material for normal postoperative metabolism. Twenty flaps showed some abnormality in the clinical course or with MD. Of these, 13 flaps were reoperated for anastomosis thrombosis (9 arterial, 4 venous). All thromboses were clearly recognized by MD via a decrease in the glucose concentration in the tissue (< 2.7 mmol/l) and an increase in the lactate concentrations (> 5.7 mmol/l). In some cases, MD indicated a pathological trend in glucose and lactate concentrations hours before there were any clinical signs. In the series, the salvage rate of all thrombosed flaps was 77%, with a final success rate in microvascular reconstruction of 95%.

In 2013, a study used microdialysis in monitoring 268 microsurgical flaps and compared the metabolic data of normally perfused flaps to those that suffered from relative or absolute perfusion failure. It was found that tissue glucose and lactate concentrations and especially their relation (lactate-to-glucose ratio) can show the presence of ischemia and aid in decision making, whether to reoperate or not. High lactate-to-glucose ratio may also predict total or partial flap necrosis. If L/G ratio was 1.0, the flap was safe. If the level reached 5.0, still only 8% suffered from some kind of difficulty but when the level of 22 was reached, the ischemia was usually evident. [38]

Most previous studies that measured blood glucose and lactate values inside the flap tissue used microdialysis.[31,39-42] Microdialysis is a method used during surgery in which a catheter is embedded in the flap tissue (eg, skin, muscle, or fat) to measure the target substance. This allows for continuous measurement and excellent objectivity, but the

complicated procedure and need for professional management have been cited as having disadvantages.[40]

By contrast, a study by Sakakibara et al examined blood glucose values in free flaps with a simple measuring instrument.[29]

In 2014, Henault et al.[43] reported a study in which measurements of capillary glucose and lactate in the flaps were done. These parameters were compared to standardized clinical monitoring during the first five days. Two sets of data (eventful versus uneventful postoperative period) were analyzed to define the thresholds of lactate and glucose values for diagnosis of pedicle complications, and to establish parameters for this screening test. Over a period of 19 months, 37 patients were included. With 5 pedicle impairments, complication thresholds were defined as lactate >= 6.4 mmol/L and glucose<= 3.85 mmol/L, in order to obtain a sensitivity of 98.5% and a specificity of 99.5% for the test. Modifications of capillary glucose and lactate measurements appeared in average 5.7 hours earlier than clinical symptoms in pedicle impairments. The mean cost of a five-day monitoring was about 90 USD. Thus, they concluded that this simple and cheap technique could be used as a routine technique in monitoring free flaps to improve safety of this reconstructive technique.

Similar study aimed by Yuma Kobayashi et al in 2022 [44], to detect early stage venous congestion in free flaps performed for reconstruction of extensive soft tissue defect following Gustilo-Anderson type IIIB and IIIC open fractures of lower limbs, included 22 free flaps in their study and used pinprick test to measure blood glucose and lactate levels. In the 7 congested limbs, the mean flap blood glucose level immediately before salvage surgery was 3.8 ± 2.0 (7.4–1.8) mmol/L. The ratio to blood glucose levels in healthy fingertips was 0.6 ± 0.2 (0.8–0.3). Lactate levels increased to 13.3 ± 5.1 (9.4–22.8) mmol/L. The ratio of blood glucose levels in congested flaps and in healthy fingertips was significantly lower than that in non-congested flaps and in healthy fingertips ($p = 0.0016$). Lactate levels were significantly higher in patients with congestion

($p = 0.0013$). Thus, they concluded that flap blood glucose and lactate levels provide a quantitative method of evaluating blood flow and detecting flow abnormalities postoperatively, and are useful in detecting early congestion due to venous thrombosis.

In 2019, Keith Kishi et al [45] prospectively analyzed 82 cases of head and neck cancer reconstruction of which 74 had impeded blood flow. Glucose and lactate levels were regularly measured over a period of 48 hours, from the time of flap elevation, as predictor variables. They observed that the intra-flap blood glucose values in the normally progressing cases gradually decreased until 16 hours postoperatively and thereafter recovered to normal levels whereas the Intra-flap blood lactate values increased until 8 hours postoperatively and subsequently decreased. The odds ratio during congestion was only significantly different for lactate (odds ratio, 2.55, $P = .014$), and the cutoff values for sensitivity and specificity were 4.2 mmol/L and 6.7 mmol/L, respectively, concluding that Intra-flap blood glucose and lactate values may reflect the transition of the postoperative circulation of free flaps. Also, during congestion, lactate values change more sensitively than blood glucose values.

The aim of our study was to compare the clinical assessment and intracapillary glucose and lactate measurements along with their ratios (L/G) during monitoring of the flaps and find out which helps in early salvage of the flaps. This method of assessment of flap viability is simple, cost effective and requires little special equipment or learning curve.

MATERIAL & METHODS

A Prospective study was conducted on all consenting patients who underwent pedicled flap or free flap surgery in the Department of Plastic Surgery, Dayanand Medical College and Hospital, Ludhiana from August 2021 to December 2022.

METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA:

- All the consenting patients undergoing any kind of flap surgery having accessible skin paddle.

EXCLUSION CRITERIA:

- Patients who did not give consent.
- Flaps that did not bleed on a pin prick.
- Flaps with inaccessible skin paddle.
- All diabetic patients.
- All critically ill patients.

The surgical procedure for flap surgery was performed. At the completion of surgery, the measurements were done for capillary glucose and capillary lactate levels from the distal 1/3rd of the flap using 2 devices: one for capillary glucose (Freestyle Optium Neo H) and other for capillary lactate (Lactate Scout 4). The values were recorded in mmol/l and then the capillary lactate to glucose ratio (L/G) was calculated every time.

Measurements was done at 0,1,3,6,12,24 and 48hrs after surgery, where 0 is immediately post operatively.

Along with L/G ratio (LGR) measurements clinical evaluation of the flap was also recorded at same 0,1,3,6,12,24 and 48 hrs which included skin paddle colour, capillary refill, temperature and bleeding on pin prick. Clinical outcome of flap was also recorded. The individual variable was classified as:

- Colour (Same as surrounding-SAS / Bluish),

- Capillary refill (within normal limit-WNL [1-3sec] / Spontaneous [<1sec] / Delayed [>3sec] / Absent),
- Bleeding characteristics (Arterial if bright red in colour / Venous if dark red in colour / No bleed),
- Temperature (Same as Surrounding-SAS / Cold) and
- Clinical outcome of flap (Survived / Not Survived / Marginal Necrosis)

Measurements were also done whenever flap showed any signs of compromised vascularity in between these intervals.

Data was divided into 2 groups:

Group A: Flaps with no post-operative complications

Group B: Flap with post-operative complications (signs of necrosis, need of any surgical intervention)

Decision to intervene surgically was based exclusively on Clinical assessment and operating surgeon's experience.

OUTCOME

The L/G ratio (LGR) of 2 groups was compared and data was utilized to calculate the cut off value of L/G ratio at which flaps begin to show signs of non-viability.

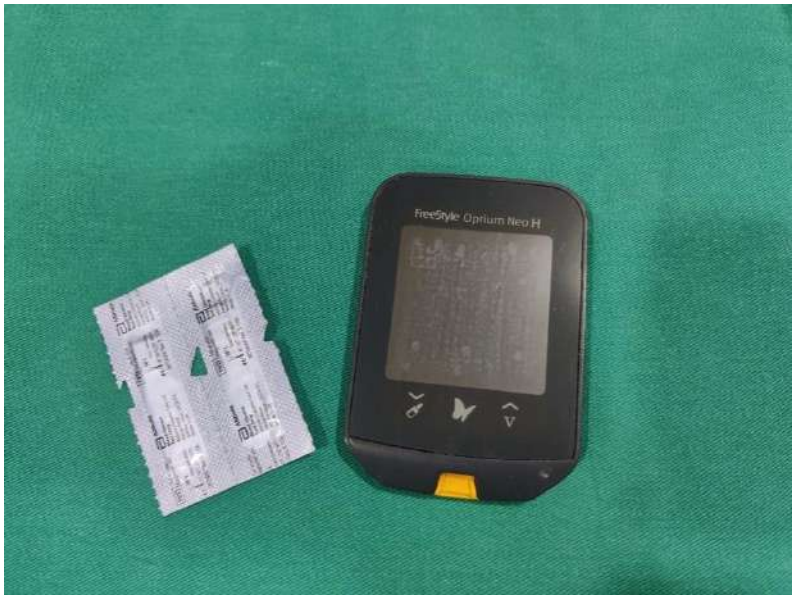
Final outcome of the flap and any intervention required was also noted.

Statistical Analysis

Data were described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. To determine whether the data were normally distributed, a Kolmogorov-Smirnov test was used. Comparison of quantitative variables between the study groups was done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test was performed, and fisher exact test was used when the expected frequency is less than 5. Receiver operator characteristics (ROC) curve was done, and criterion value was estimated depending on

the specificity and sensitivity. Area under curve (AUC) was measured. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations were done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

Picture 1: Lactometer with disposable strips



Picture 2: Glucometer with disposable strips

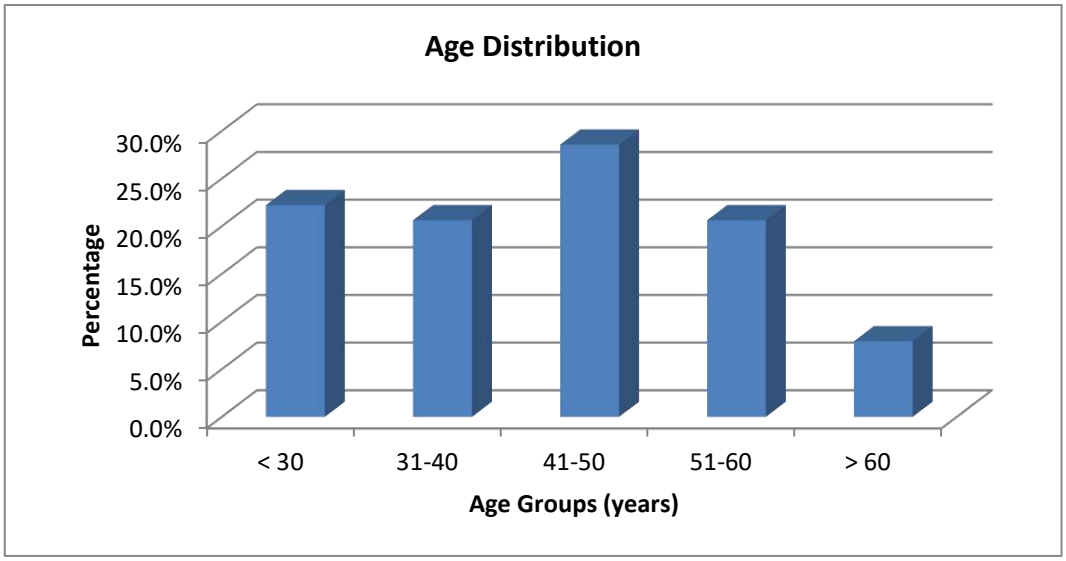
OBSERVATION & RESULTS

Over a period of 17 months (August 2021 to December 2022), 63 flaps were included in the study with minimum age of 13 years and maximum age of 74 years with mean of 42±14.5 years. Table 1 and Figure 1 shows frequency distribution of total cases (n=63) according to age with a total of 14 cases under 30 years of age, 13 cases between 31-40 years and 51-60 years each, 18 cases between 41-50 years and 5 cases above 60 years of age.

Table 1: Age distribution

Age group	No. of cases	Percentage
< 30	14	22.2%
31-40	13	20.6%
41-50	18	28.6%
51-60	13	20.6%
> 60	5	7.9%
Total	63	100.0%

Figure 1: Age distribution

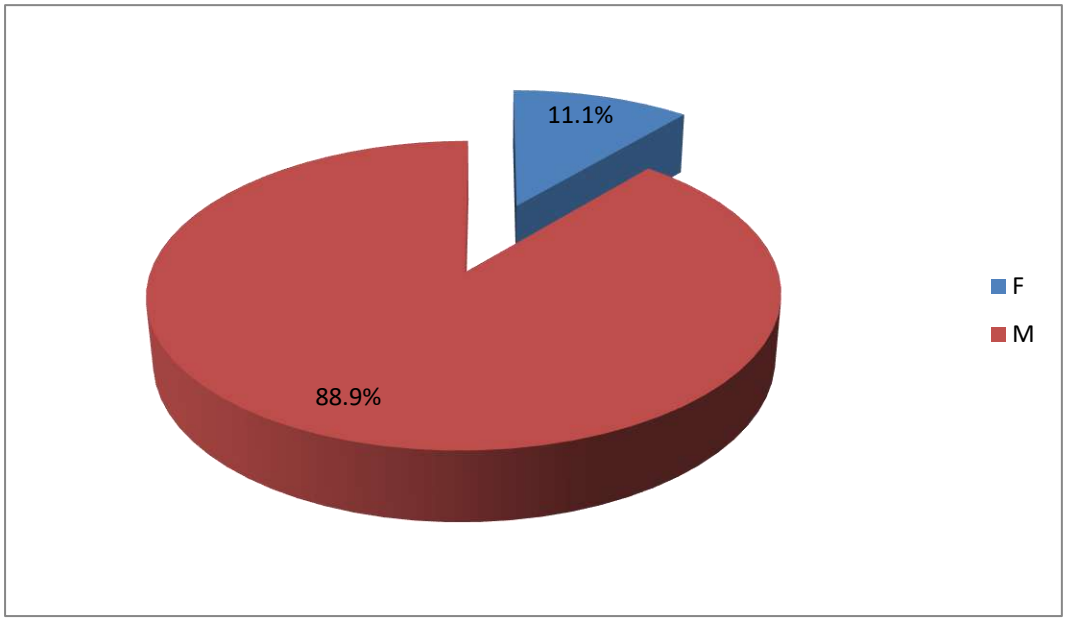


Out of 63 cases, there were 7 females (11.1%) and 56 were males (88.9%) as shown in Table 2 and Figure 2:

Table 2: Gender distribution

SEX(M/F)	No. of cases	Percentage
F	7	11.1%
M	56	88.9%
Total	63	100.0%

Figure 2: Gender distribution

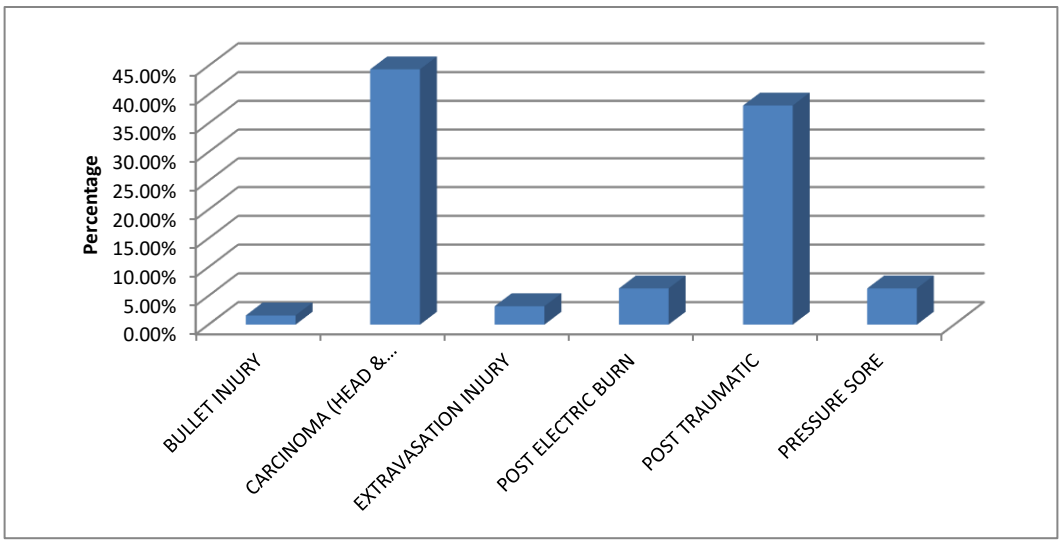


Out of 63 cases, aetiology of 25 cases (39.7%) was head and neck cancers making them the most common cause followed by trauma in 24 cases (38.1%). Other causes of defect were post electric burn (6.3%), pressure sores (6.3%), carcinomas other than head and neck cancer (4.8%), extravasation injuries (3.2%) and bullet injury (1.6%). (Table 3 & Figure 3)

Table 3: Frequency distribution of cause of Defect

CAUSE OF DEFECT	No. of cases	Percentage
Bullet injury	1	1.6%
Carcinoma (head & neck/breast/sacrum/foot)	28	44.4%
Extravasation injury	2	3.2%
Post electric burn	4	6.3%
Post traumatic	24	38.1%
Pressure sore	4	6.3%
Total	63	100.0%

Figure 3: Frequency distribution of cause of Defect

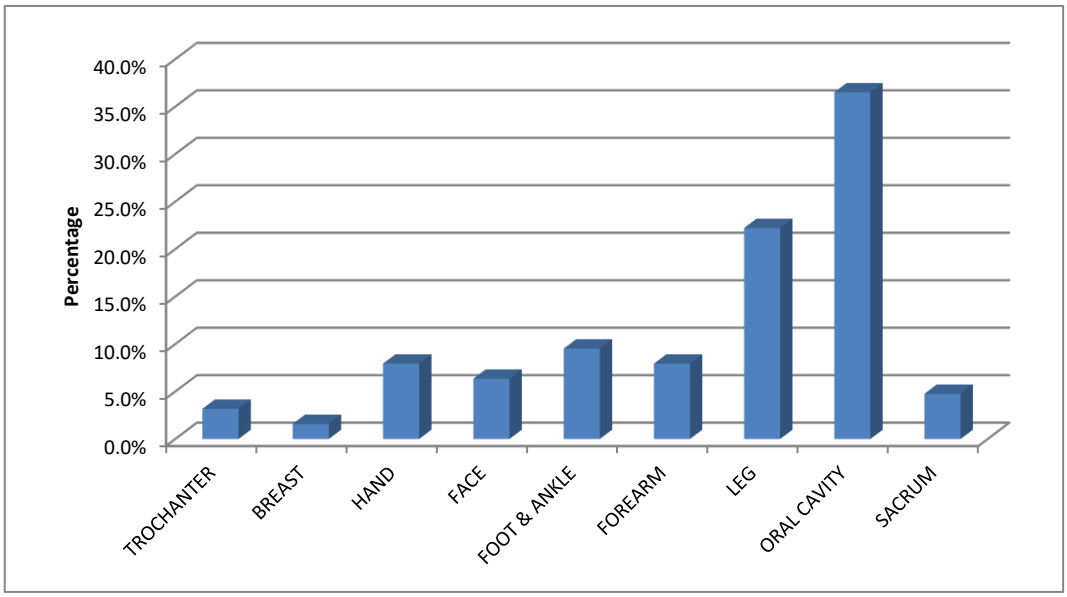


The most common site of defect was oral cavity (36.5%) followed by leg (22.2%), foot & ankle, forearm, hand, face, sacrum, trochanter and breast in the order as shown in Table 4 and Figure 4.

Table 4: Frequency distribution of wound site

WOUND SITE	No. of cases	Percentage
Trochanter	2	3.2%
Breast	1	1.6%
Hand	5	7.9%
Face	4	6.3%
Foot & ankle	6	9.5%
Forearm	5	7.9%
Leg	14	22.2%
Oral cavity	23	36.5%
Sacrum	3	4.8%
Total	63	100.0%

Figure 4: Frequency distribution of wound site

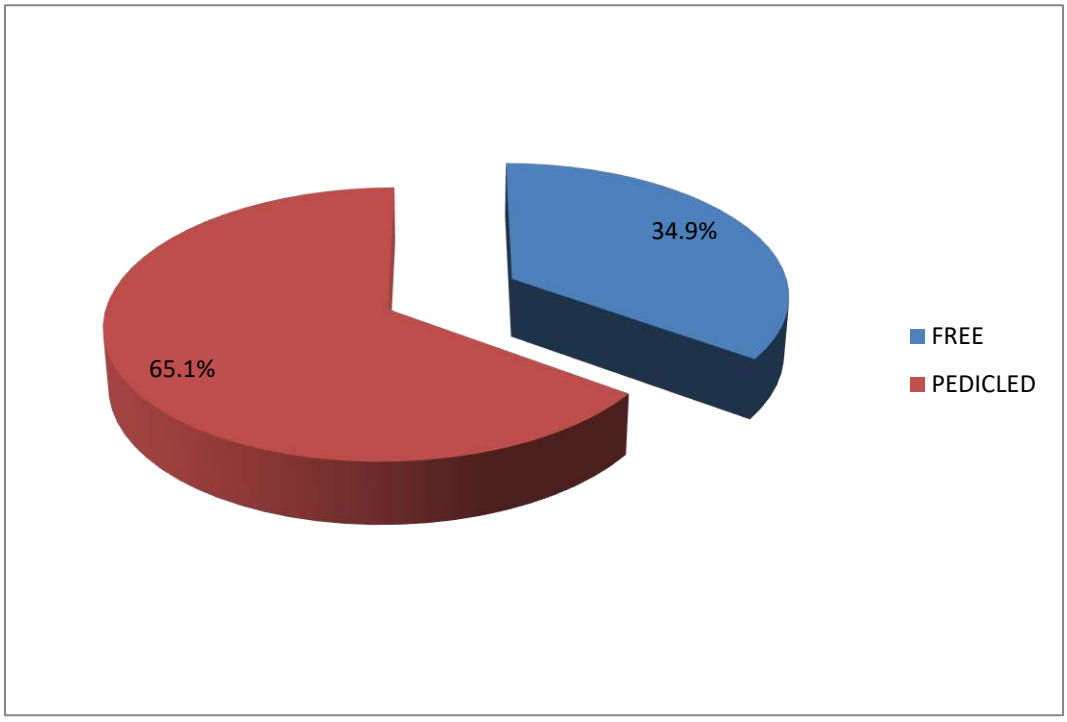


Both Free as well as pedicled flaps were included in the study. Among total cases, 22 of 63 were free flaps and 41 of 63 were Pedicled flaps. (Table 5, Figure 5)

Table 5: Types of Flaps

TYPE OF FLAPS	No. of cases	Percentage
Free	22	34.9%
Pedicled	41	65.1%
Total	63	100.0%

Figure 5: Types of Flaps

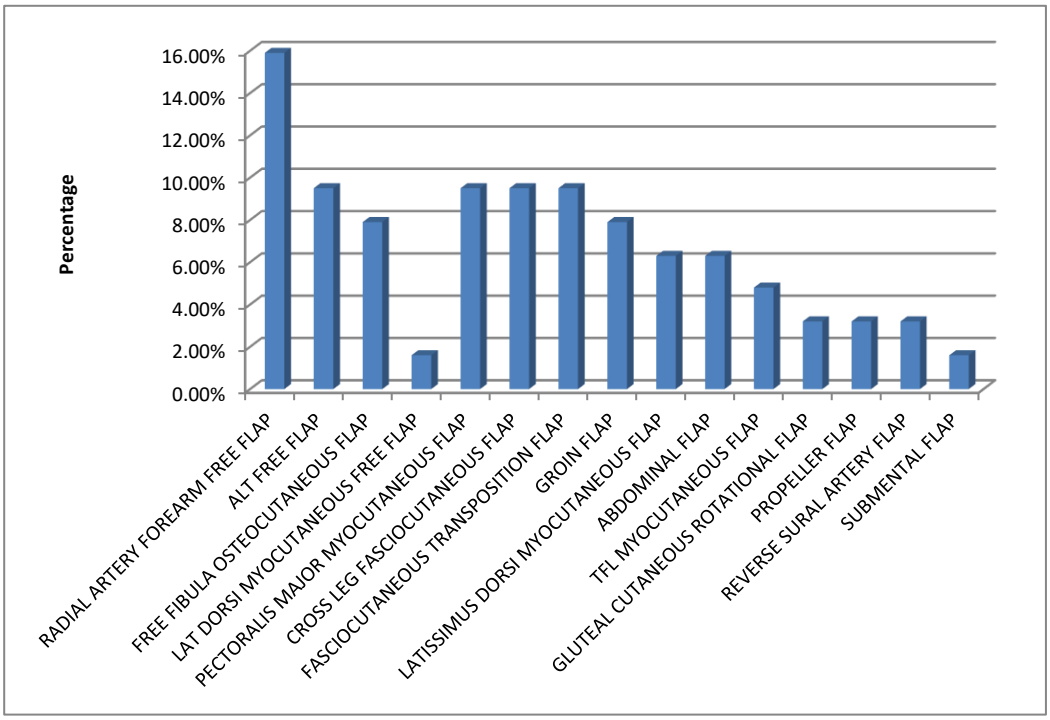


Among Free Flaps, majority were Radial Artery Forearm Free Flap (15.9%) followed by ALT free flap (9.5%), Free fibula Osteocutaneous flap (7.9%) and LD Free flap (1.6%) and the Pedicled flaps included PMMC, Transposition flap, Cross leg Fasciocutaneous flap, LD myocutaneous flap, TFL Flap, Abdominal Flap, Groin flap, Gluteal rotational flap, Reverse Sural Artery flap and Submental flap. (Table 6 & Figure 6)

Table 6: Frequency distribution of Different types of Flaps

FLAP NAME	No. of cases	Percentage
Radial artery forearm free flap	10	15.9%
ALT free flap	6	9.5%
Free Fibula osteocutaneous flap	5	7.9%
LatissimusDorsi myocutaneous free flap	1	1.6%
Pectoralis Major myocutaneous flap	6	9.5%
Cross leg fasciocutaneous flap	6	9.5%
Fasciocutaneous transposition flap	6	9.5%
Groin flap	5	7.9%
Latissimus Dorsi myocutaneous flap	4	6.3%
Abdominal flap	4	6.3%
TFL myocutaneous flap	3	4.8%
Gluteal cutaneous rotational flap	2	3.2%
Propeller flap	2	3.2%
Reverse Sural artery flap	2	3.2%
Submental flap	1	1.6%
Total	63	100.0%

Figure 6: Frequency distribution of Different types of Flaps



Data was divided into two groups –

- Group A (Flap without post operative complications) &
- Group B (Flap with complications- non survival, marginal necrosis or those that needed surgical intervention as salvage);

Both groups were statistically compared on the basis of demographic details of the patients as well as other parameters and p value was calculated.

On comparing both groups in terms of age and gender distribution and applying chi-square test, P-value was found to be non-significant depicting that age as well as gender is not related to complications in flaps. (Table 7, Figure 7 for age) & (table 8, Figure 8 for gender)

Table 7: Comparison of age distribution in both groups

		Flap without complication (GROUP A) N=51		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Age group	< 30	9	64.3%	5	35.7%	14	7.095	0.131
	31-40	9	69.2%	4	30.8%	13		
	41-50	17	94.4%	1	5.6%	18		
	51-60	11	84.6%	2	15.4%	13		
	> 60	5	100.0%	0	0.0%	5		
Total		51	81.0%	12	19.0%	63		

Figure 7: Comparison of age distribution in both groups

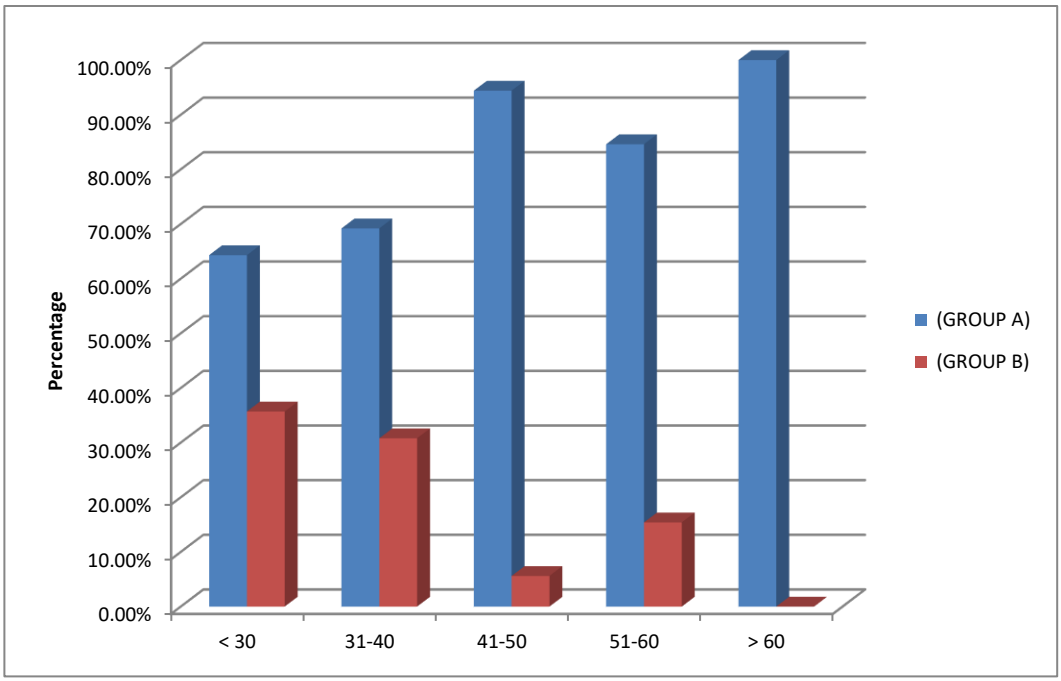
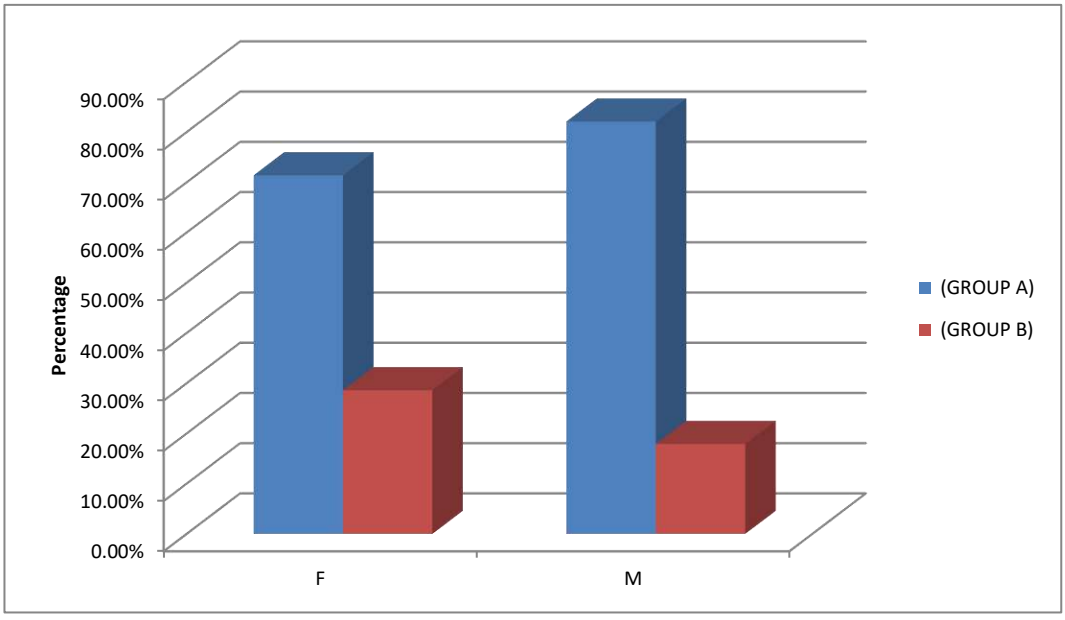


Table 8: Comparison of gender distribution in both groups

		Flap without complication (GROUP A) N=51		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
SEX(M/F)	F	5	71.4%	2	28.6%	7	0.463	0.496
	M	46	82.1%	10	17.9%	56		
Total		51	81.0%	12	19.0%	63		

Figure 8: Comparison of gender distribution in both groups

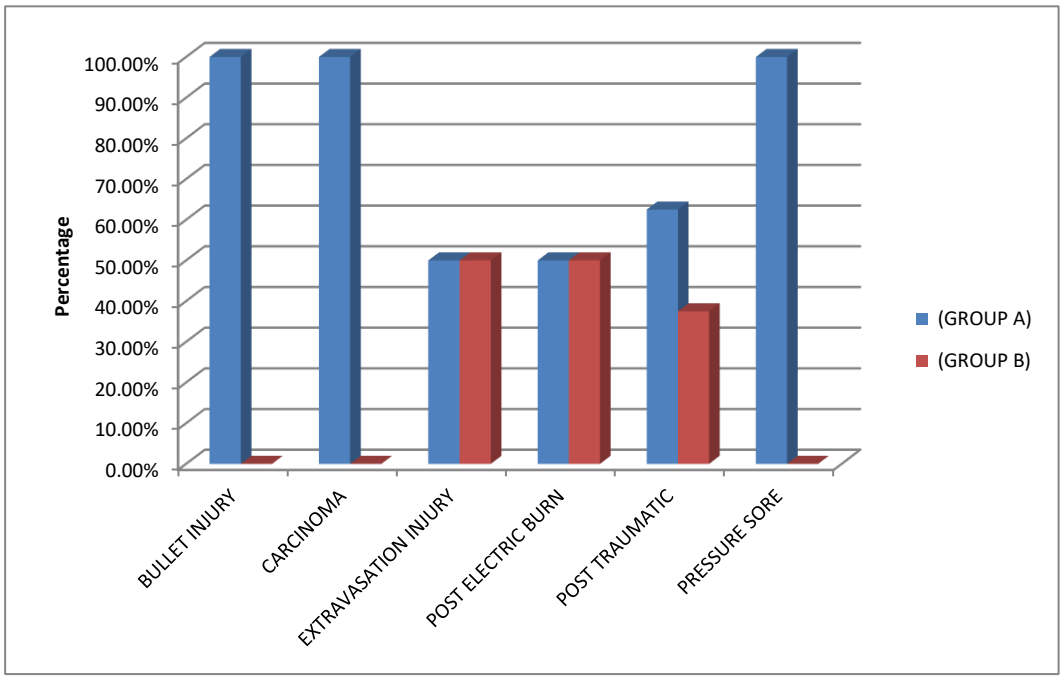


On the other hand, etiology of the defect as well as site of the defect were significantly associated with complications or viability of flaps. Table 9 & Figure 9 shows that 9 out of 24 post traumatic cases (37.5%) had complications with flaps whereas none of the flaps with Carcinoma as the etiology developed complications.

Table 9: Comparison of cause of defect in both groups

		Flap without complication (GROUP A) N=51		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
CAUSE OF DEFECT	Bullet injury	1	100.0%	0	0.0%	1	16.792	0.010
	Carcinoma	28	100.0%	0	0.0%	28		
	Extravasation injury	1	50.0%	1	50.0%	2		
	Post electric burn	2	50.0%	2	50.0%	4		
	Post traumatic	15	62.5%	9	37.5%	24		
	Pressure sore	4	100.0%	0	0.0%	4		
Total		51	81.0%	12	19.0%	63		

Figure 9: Comparison of cause of defect in both groups

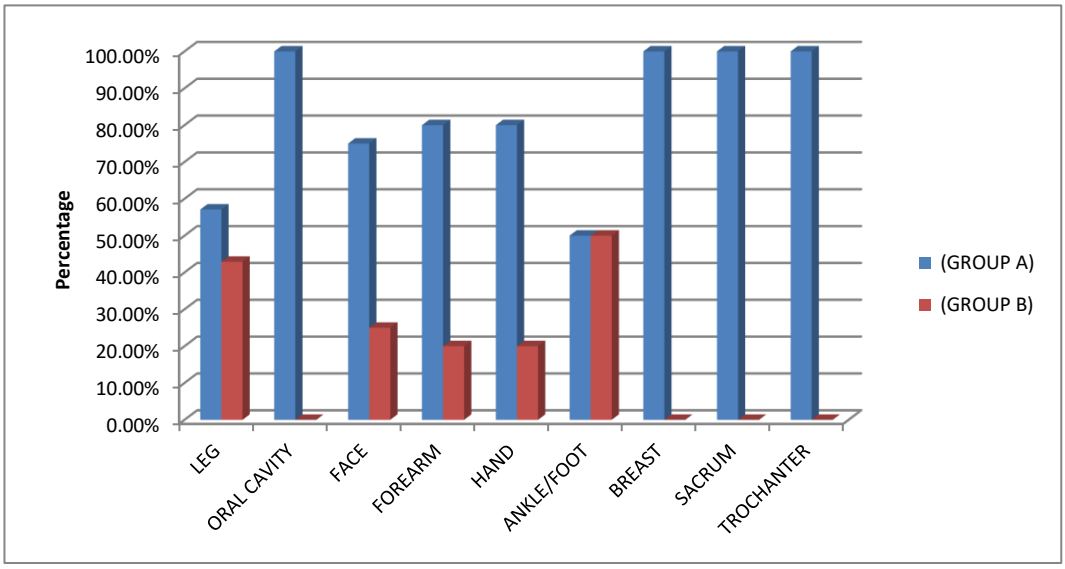


Similarly, 6 out of 14 cases (42.9%) where leg was the defect site developed complications whereas none of the flaps used to cover oral cavity defect showed complications. Thus, the wound or defect site was found to be significantly associated with flap viability. (Table 10 & Figure 10)

Table 10: Comparison of Wound site in both groups

		Flap without complication (GROUP A) N=51		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Wound site	Leg	8	57.1%	6	42.9%	14	15.796	0.045
	Oral cavity	23	100.0%	0	0.0%	23		
	Face	3	75.0%	1	25.0%	4		
	Forearm	4	80.0%	1	20.0%	5		
	Hand	4	80.0%	1	20.0%	5		
	Ankle/foot	3	50.0%	3	50.0%	6		
	Breast	1	100.0%	0	0.0%	1		
	Sacrum	3	100.0%	0	0.0%	3		
	Trochanter	2	100.0%	0	0.0%	2		
Total		51	81.0%	12	19.0%	63		

Figure 10: Comparison of Wound site in both groups

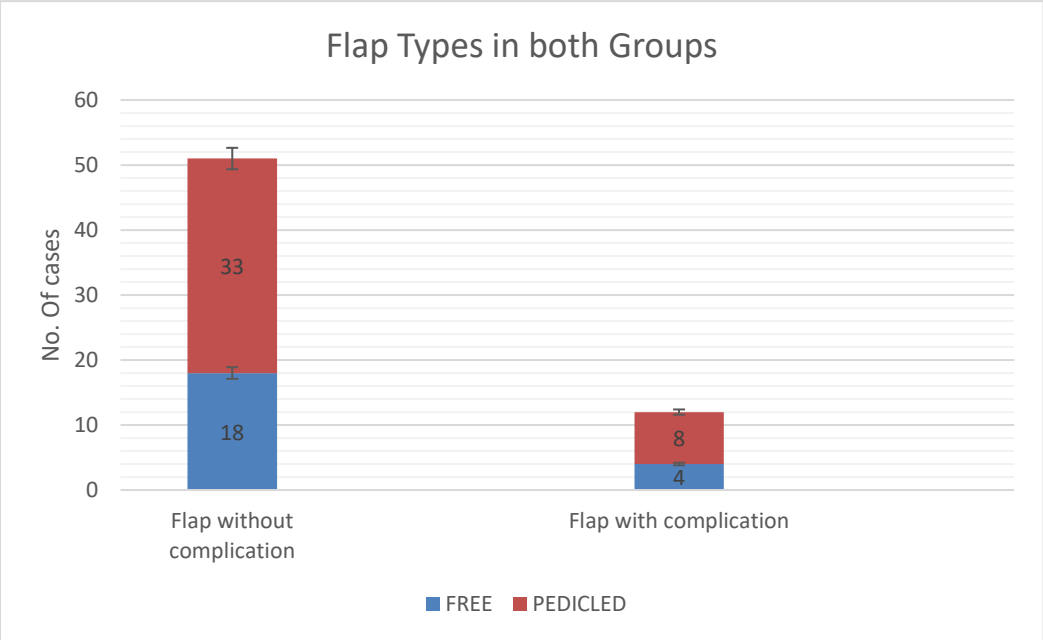


4 out of 22 free flaps (18.2%) and 8 out of 33 pedicled flaps (19.5%) developed complications. On comparing both the groups in terms of Flap Type, the P-value came out to be insignificant, demonstrating that free or pedicled flaps were not related to flap viability or associated complications. (Table 11 & Figure 11)

Table 11: Comparison of Flap type in both groups

		Flap without complication (GROUP A) N=51		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Flap type	Free	18	81.8%	4	18.2%	22	0.016	0.898
	Pedicled	33	80.5%	8	19.5%	41		
Total		51	81.0%	12	19.0%	63		

Figure 11: Comparison of Flap type in both groups



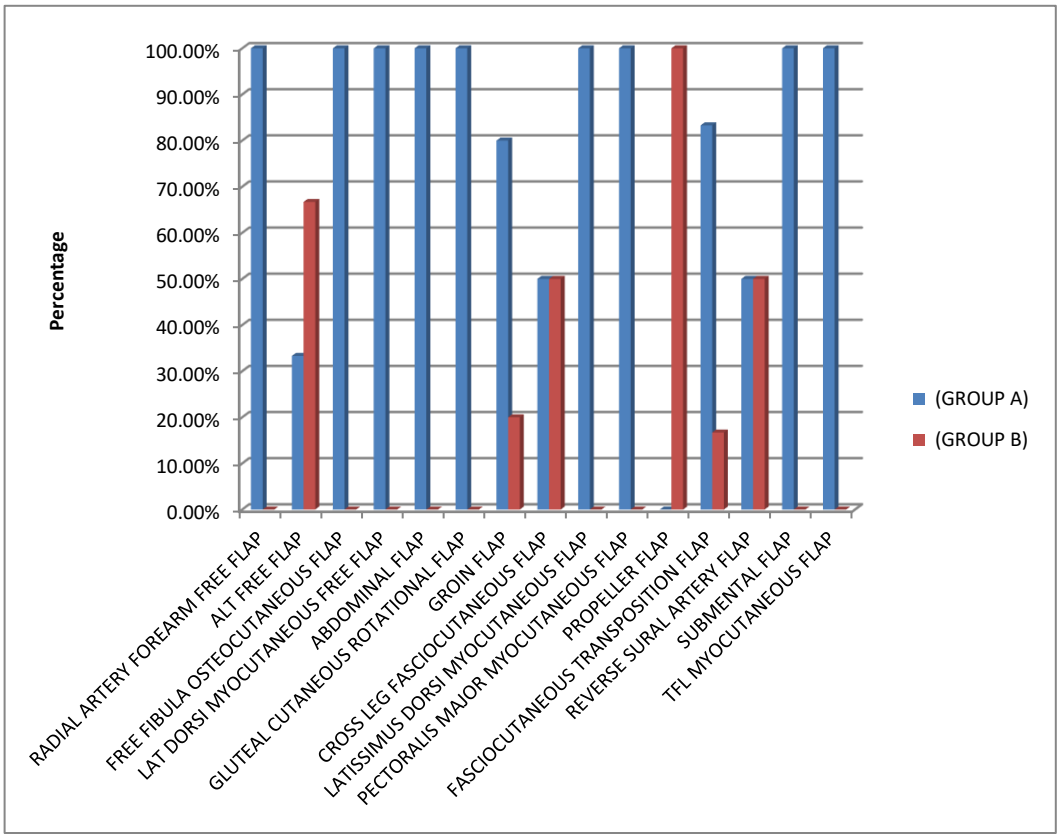
FLAP NAME

Group B i.e. Flap with complications; comprised total of 12/63 cases with majority being ALT free flap (33.3%), followed by Cross leg Fasciocutaneous flaps (25%), propeller flap (16.7%), RSA flap, groin flap and Fasciocutaneous transposition flap (8.3% each). Among the free flaps that developed complications, all of the 4 were ALT free flaps whereas among Pedicled flaps, both the propeller flaps (100%), 50% of Cross leg Fasciocutaneous flaps and 50% of Reverse Sural Artery Flap developed complications. (Table 12 & Figure 12)

TABLE 12: Comparison of Flap name in both groups

		Flap without complication (GROUP A) N=51		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Flap Name	Radial artery forearm free flap	10	100.0%	0	0.0%	10	30.79	0.009
	ALT free flap	2	33.3%	4	66.7%	6		
	Free Fibula osteocutaneous flap	5	100.0%	0	0.0%	5		
	Latissimus Dorsi myocutaneous free flap	1	100.0%	0	0.0%	1		
	Abdominal flap	4	100.0%	0	0.0%	4		
	Gluteal cutaneous rotational flap	2	100.0%	0	0.0%	2		
	Groin flap	4	80.0%	1	20.0%	5		
	Cross leg fasciocutaneous flap	3	50.0%	3	50.0%	6		
	Latissimus Dorsi myocutaneous flap	4	100.0%	0	0.0%	4		
	Pectoralis Major myocutaneous flap	6	100.0%	0	0.0%	6		
	Propeller flap	0	0.0%	2	100.0%	2		
	Fasciocutaneous transposition flap	5	83.3%	1	16.7%	6		
	Reverse Sural artery flap	1	50.0%	1	50.0%	2		
	Submental flap	1	100.0%	0	0.0%	1		
	TFL myocutaneous flap	3	100.0%	0	0.0%	3		
Total		51	81.0%	12	19.0%	63		

Figure 12**:** Comparison of Flap name in both groups



CLINICAL MONITORING OF FLAPS.

All the flaps were clinically monitored and the observations in terms of

- Colour (Same as surrounding-SAS or Bluish),
- Capillary refill (within normal limit-WNL[1-3sec], Spontaneous [<1sec], Delayed[>3sec] or Absent),
- Bleeding characteristics (Arterial if bright red in colour, venous if dark red in colour or No bleed),
- Temperature (Same as Surrounding-SAS or Cold) and
- Clinical outcome of flap (Survived, Not Survived or Marginal Necrosis)

were recorded at several intervals (0 hrs, 1 hr, 3 hrs, 6 hrs, 12 hrs, 24 hrs and 48 hrs). All these clinical findings were compared in both groups and p-value was calculated.

At 0 hrs, P-value was found to be non-significant i.e., there was no statistical difference in both groups and all flaps survived. (Table 13)

Table 13: Clinical monitoring of flaps of both groups at 0 hours

At 0 hours		**Flap without complication (GROUP A) N=51**		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Colour	SAS	51	100.0%	12	100.0%	63		
Capillary refill	SPONTANEOUS	1	2.0%	1	8.3%	2	1.283	0.347
	WNL	50	98.0%	11	91.7%	61		
Bleeding characteristics	ARTERIAL	50	98.0%	12	100.0%	62	0.239	0.625
	VENOUS	1	2.0%	0	0.0%	1		
Temperature	SAS	51	100.0%	12	100.0%	63		
Outcome of flap	SURVIVED	51	100.0%	12	100.0%	63		

At 1 hrs, the color and bleeding characteristics showed statistically significant difference in both the groups and 1 flap developed complications. (Table 14)

Table 14: Clinical monitoring of flaps of both groups at 1 hours

At 1 hrs		**Flap without complication (GROUP A) N=51**		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Colour	BLUISH	0	0.0%	2	16.7%	2	8.779	0.034
	SAS	51	100.0%	10	83.3%	61		
Capillary refill	SPONTANEOUS	1	2.0%	2	16.7%	3	4.632	0.09
	WNL	50	98.0%	10	83.3%	60		
Bleeding characteristics	ARTERIAL	51	100.0%	9	75.0%	60	13.388	0.006
	VENOUS	0	0.0%	3	25.0%	3		
Temperature	COLD	0	0.0%	1	8.3%	1	4.319	0.190
	SAS	51	100.0%	11	91.7%	62		
Outcome of flap	MARGINAL NECROSIS	0	0.0%	1	8.3%	1	4.319	0.190
	SURVIVED	51	100.0%	11	91.7%	62		

At 3 hours, both groups showed statistical difference in terms of colour, capillary refill and bleeding characteristics with P-value <0.05. (Table 15)

Table 15: Clinical monitoring of flaps of both groups at 3 hours

At 3 hrs		**Flap without complication (GROUP A) N=51**		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Colour	BLUISH	0	0.0%	2	16.7%	2	8.779	0.034
	SAS	51	100.0%	10	83.3%	61		
Capillary refill	ABSENT	0	0.0%	1	8.3%	1	13.388	0.001
	DELAYED	0	0.0%	2	16.7%	2		
	WNL	51	100.0%	9	75.0%	60		
Bleeding characteristics	ARTERIAL	49	96.1%	7	58.3%	56	15.496	0.001
	NO BLEED	0	0.0%	2	16.7%	2		
	VENOUS	2	3.9%	3	25.0%	5		
Temperature	COLD	0	0.0%	1	8.3%	1	4.319	0.190
	SAS	51	100.0%	11	91.7%	62		
Outcome of flap	MARGINAL NECROSIS	0	0.0%	1	8.3%	1	4.319	0.190
	SURVIVED	51	100.0%	11	91.7%	62		

At 6, 12, 24 and 48 hrs there was observed a statistically significant difference in both the groups in term as of all variables.(Table 16, 17, 18, 19)

Table 16: Clinical Monitoring of Flaps of both groups at 6 Hours

At 6 hrs		**Flap without complication (GROUP A) N=51**		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Colour	BLUISH	0	0.0%	3	25.0%	3	23.082	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	2	16.7%	2		
	SAS	51	100.0%	7	58.3%	58		
Capillary refill	DELAYED	0	0.0%	2	16.7%	2	23.082	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	2	16.7%	2		
	SPONTANEOUS	0	0.0%	1	8.3%	1		
	WNL	51	100.0%	7	58.3%	58		
Bleeding characteristics	ARTERIAL	50	98.0%	4	33.3%	54	33.422	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	2	16.7%	2		
	VENOUS	1	2.0%	6	50.0%	7		
Temperature	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	2	16.7%	2	8.779	0.034
	SAS	51	100.0%	10	83.3%	61		
Outcome of flap	MARGINAL NECROSIS	0	0.0%	2	16.7%	2	8.779	0.034
	SURVIVED	51	100.0%	10	83.3%	61		

Table 17: Clinical monitoring of flaps of both groups at 12 hours

At 12 hrs		**Flap without complication (GROUP A) N=51**		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Colour	BLUISH	0	0.0%	4	33.3%	4	33.469	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	3	25.0%	3		
	SAS	51	100.0%	5	41.7%	56		
Capillary refill	ABSENT	0	0.0%	1	8.3%	1	28.184	0.001
	DELAYED	0	0.0%	1	8.3%	1		
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	3	25.0%	3		
	SPONTANEOUS	0	0.0%	1	8.3%	1		
	WNL	51	100.0%	6	50.0%	57		
Bleeding characteristics	ARTERIAL	50	98.0%	3	25.0%	53	39.087	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	3	25.0%	3		
	VENOUS	1	2.0%	6	50.0%	7		
Temperature	COLD	0	0.0%	1	8.3%	1	18.153	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	3	25.0%	3		
	SAS	51	100.0%	8	66.7%	59		
Outcome of flap	MARGINAL NECROSIS	0	0.0%	6	50.0%	6	28.184	0.001
	SURVIVED	51	100.0%	6	50.0%	57		

Table 18: Clinical monitoring of flaps of both groups at 24 hours

At 24 hrs		Flap without complication (GROUP A) N=51		Flap with complication (GROUP B) N=12		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Colour	BLUISH	0	0.0%	6	50.0%	6	56.639	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	SAS	51	100.0%	1	8.3%	52		
Capillary refill	ABSENT	0	0.0%	3	25.0%	3	56.639	0.001
	DELAYED	0	0.0%	2	16.7%	2		
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	SPONTANEOUS	0	0.0%	1	8.3%	1		
	WNL	51	100.0%	1	8.3%	52		
Bleeding characteristics	ARTERIAL	51	100.0%	2	16.7%	53	50.519	0.001
	NO BLEED	0	0.0%	2	16.7%	2		
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	VENOUS	0	0.0%	3	25.0%	3		
Temperature	COLD	0	0.0%	2	16.7%	2	33.469	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	SAS	51	100.0%	5	41.7%	56		
Outcome of flap at 24 hrs	MARGINAL NECROSIS	0	0.0%	9	75.0%	9	44.625	0.001
	SURVIVED	51	100.0%	3	25.0%	54		

Table 19: Clinical monitoring of flaps of both groups at 48 hours

At 48 hrs		Flap without complication (GROUP A) N=51		Flap with complication (GROUP B) N=12		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Colour	BLUISH	0	0.0%	6	50.0%	6	56.639	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	SAS	51	100.0%	1	8.3%	52		
Capillary refill	ABSENT	0	0.0%	3	25.0%	3	56.639	0.001
	DELAYED	0	0.0%	2	16.7%	2		
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	SPONTANEOUS	0	0.0%	1	8.3%	1		
	WNL	51	100.0%	1	8.3%	52		
Bleeding characteristics	ARTERIAL	51	100.0%	1	8.3%	52	56.639	0.001
	NO BLEED	0	0.0%	3	25.0%	3		
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	VENOUS	0	0.0%	3	25.0%	3		
Temperature	COLD	0	0.0%	2	16.7%	2	33.469	0.001
	NOT SURVIVED/MARGINAL NECROSIS	0	0.0%	5	41.7%	5		
	SAS	51	100.0%	5	41.7%	56		
Outcome of flap	MARGINAL NECROSIS	0	0.0%	11	91.7%	11	56.639	0.001
	SURVIVED	51	100.0%	1	8.3%	52		

Analysing the clinical outcome of flaps depending upon the four variables, and comparing it between both groups, it was observed that the outcome of flap at and after 6 hours shows significant difference with P-value <0.05 (table 20) with marginal necrosis present in 11 out of 12 flaps of group B by the end of 48 hours and 1 out of 12 flaps of group B survived well.

Table 20: Outcome of flap of both groups on basis of clinical examination

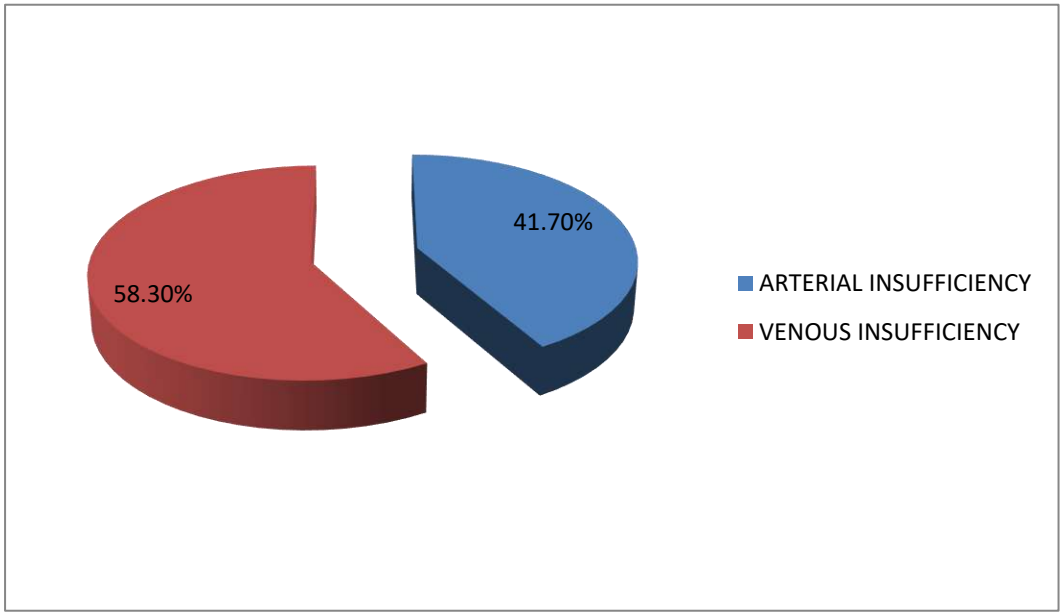
OUTCOME OF FLAP		**Flap without complication (GROUP A) N=51**		**Flap with complication (GROUP B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
AT 0 HRS	SURVIVED	51	100.0%	12	100.0%	63		
AT 1 HRS	MARGINAL NECROSIS	0	0.0%	1	8.3%	1	4.319	0.190
	SURVIVED	51	100.0%	11	91.7%	62		
AT 3 HRS	MARGINAL NECROSIS	0	0.0%	1	8.3%	1	4.319	0.190
	SURVIVED	51	100.0%	11	91.7%	62		
AT 6 HRS	MARGINAL NECROSIS	0	0.0%	2	16.7%	2	8.779	0.034
	SURVIVED	51	100.0%	10	83.3%	61		
AT 12 HRS	MARGINAL NECROSIS	0	0.0%	6	50.0%	6	28.184	0.001
	SURVIVED	51	100.0%	6	50.0%	57		
AT 24 HRS	MARGINAL NECROSIS	0	0.0%	9	75.0%	9	44.625	0.001
	SURVIVED	51	100.0%	3	25.0%	54		
AT 48 HRS	MARGINAL NECROSIS	0	0.0%	11	91.7%	11	56.639	0.001
	SURVIVED	51	100.0%	1	8.3%	52		

And among the 12 flaps in Group B (that developed complications), 7 flaps (58.3% were found to have venous insufficiency as the cause of complications and 5 flaps (41.7%) were observed having arterial insufficiency as the causative factor. (Table 21 & Figure 13)

TABLE 21: Outcome of flaps in group B in terms of arterial and venous insufficiency

OUTCOME OF FLAP	No. of cases	Percentage
ARTERIAL INSUFFICIENCY	5	41.7%
VENOUS INSUFFICIENCY	7	58.3%
Total	12	100.0%

Figure 13: Outcome of flaps in group B in terms of arterial and venous insufficiency



CAPILLARY LACTATE LEVELS

Along with clinically monitoring recorded at various intervals, the capillary lactate and glucose levels were also recorded by pricking the distal 1/3rd of the pedicled flap and margin of the skin paddle of the free flap. And the L/G Ratio (LGR) was calculated and recorded.

Table 22 describes the minimum and maximum capillary lactate values at interval 0, 1, 3, 6, 12, 24 and 48 hours along with the mean at each above said intervals.

Table 22: Mean of Capillary Lactate values at different intervals. N denotes no. of flaps.

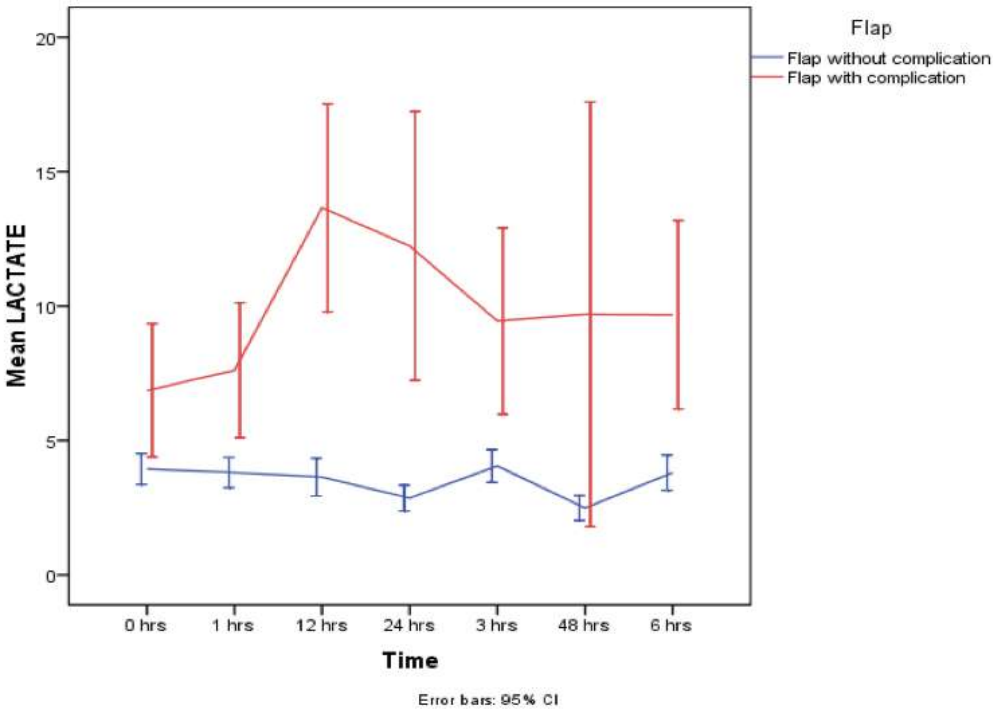
	N	Minimum	Maximum	Mean	SD
LACTATE AT 0 HRS	63	1.60	15.40	4.50	2.72
LACTATE AT 1 HR	63	1.01	17.60	4.54	2.87
LACTATE AT 3HRS	63	1.60	18.20	5.08	3.69
LACTATE AT 6HRS	61	1.10	19.90	4.77	3.59
LACTATE AT 12 HRS	59	0.60	18.20	5.00	4.47
LACTATE AT 24 HRS	56	0.40	18.20	3.70	3.33
LACTATE AT 48 HRS	55	0.60	14.40	3.02	2.71

Table 23 and Figure 14 shows comparison of both groups in terms of mean value of lactate at various intervals and there is observed a significant statistical difference with P-value <0.05; at all intervals.

Table 23: Comparison of both groups in terms of capillary lactate values at individual interval

	Flap without complication (GROUP A) N=51		**Flap with complication (GROUP B) N=12**		Z	p-value
	Mean	SD	Mean	SD		
LACTATE AT 0 HRS	3.94	2.04	6.87	3.90	-3.673	0.001
LACTATE AT 1 HR	3.81	1.99	7.62	3.95	-4.818	0.001
LACTATE AT 3HRS	4.06	2.17	9.45	5.45	-5.539	0.001
LACTATE AT 6HRS	3.81	2.32	9.68	4.91	-5.912	0.001
LACTATE AT 12 HRS	3.65	2.51	13.66	4.63	-9.229	0.001
LACTATE AT 24 HRS	2.86	1.70	12.24	4.03	-10.151	0.001
LACTATE AT 48 HRS	2.49	1.62	9.70	4.97	-7.054	0.001

Figure 14: Mean Capillary Lactate at individual intervals for both Groups



CAPILLARY GLUCOSE LEVELS

Table 24 shows the minimum and maximum capillary glucose values at interval 0, 1, 3, 6, 12, 24 and 48 hours along with the mean at each above said intervals.

Table 24: Mean of Capillary Glucose values at different intervals. N denotes no. of flaps.

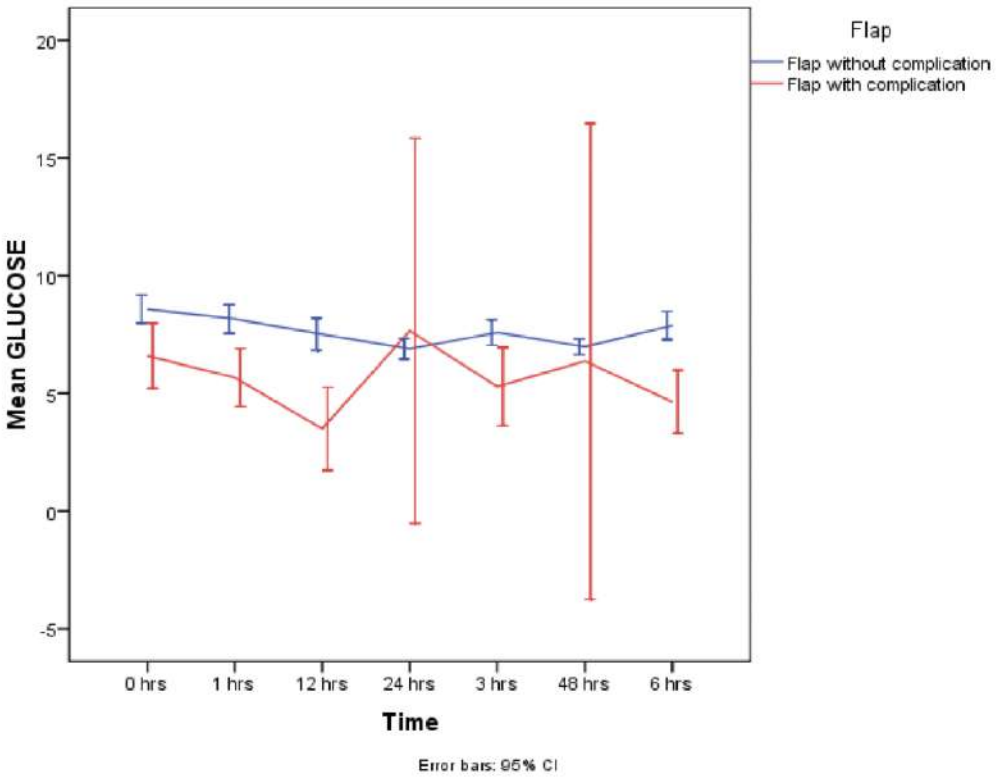
	N	Minimum	Maximum	Mean	SD
GLUCOSE AT 0 HR	63	1.50	15.21	8.20	2.27
GLUCOSE AT 1 HR	63	1.11	15.60	7.69	2.33
GLUCOSE AT 3 HRS	62	1.11	14.93	7.18	2.19
GLUCOSE AT 6 HRS	61	1.17	19.37	7.35	2.41
GLUCOSE AT 12 HRS	59	1.39	20.81	6.97	2.73
GLUCOSE AT 24 HRS	56	0.05	19.31	6.96	2.31
GLUCOSE AT 48 HRS	55	1.11	15.60	6.94	1.89

Table 25 and Figure 15 shows comparison of both groups in terms of mean value of glucose at various intervals and there is observed a significant statistical difference with P-value <0.05; at 0, 1, 3, 6 and 12 hours intervals. Whereas there appears no significant difference in mean glucose of both groups at 24 hrs and 48 hrs.

TABLE 25: Comparison of both groups in terms of capillary glucose values at individual interval.

	Flap without complication (Group A) N=51		**Flap with complication (GROUP B) N=12**		Z	p-value
	Mean	SD	Mean	SD		
GLUCOSE AT 0 HR	8.58	2.14	6.60	2.18	2.884	0.005
GLUCOSE AT 1 HR	8.16	2.17	5.67	1.94	3.648	0.001
GLUCOSE AT 3 HRS	7.59	1.91	5.28	2.47	3.435	0.001
GLUCOSE AT 6 HRS	7.89	2.14	4.64	1.87	4.469	0.001
GLUCOSE AT 12 HRS	7.51	2.41	3.50	2.11	4.447	0.001
GLUCOSE AT 24 HRS	6.89	1.54	7.66	6.59	-0.703	0.485
GLUCOSE AT 48 HRS	6.98	1.19	6.38	6.36	0.614	0.542

Figure 15: Mean Capillary Glucose at individual intervals for both Groups



CAPILLARY LACTATE/GLUCOSE RATIO

Table 26 shows the minimum and maximum capillary lactate / glucose ratio at interval 0, 1, 3, 6, 12, 24 and 48 hours along with the mean at each above said intervals.

TABLE 26: Mean of Capillary Lactate / Glucose ratio (LGR) at different intervals. N denotes no. of flaps.

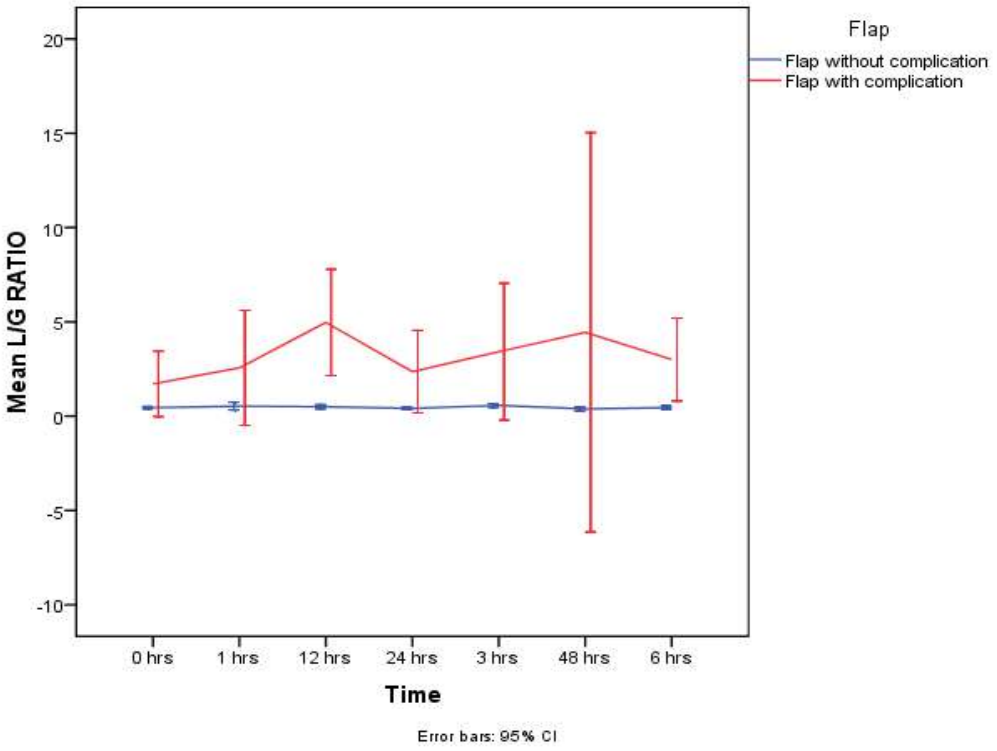
	N	Minimum	Maximum	Mean	SD
L/G RATIO AT 0 HRS	63	0.03	10.20	0.69	1.27
L/G RATIO AT 1 HRS	63	0.02	17.60	0.92	2.27
L/G RATIO AT 3 HRS	62	0.18	18.20	1.07	2.47
L/G RATIO AT 6 HRS	61	0.02	10.77	0.88	1.55
L/G RATIO AT 12 HRS	59	0.05	11.80	1.10	1.97
L/G RATIO AT 24 HRS	56	0.06	5.29	0.59	0.77
L/G RATIO AT 48 HRS	55	0.10	14.40	0.68	1.92

Table 27 and Figure 16 shows comparison of both groups in terms of mean value of Capillary Lactate / Glucose ratio at various intervals and there is observed a significant statistical difference with P-value <0.05; at 0, 1, 3, 6, 12, 24 and 48 hours intervals.

TABLE 27: Comparison of both groups in terms of capillary lactate/ glucose ratio (LGR) at individual interval.

	Flap without complication (Group A) N=51		**Flap with complication (GROUP B) N=12**		Z	p-value
	Mean	SD	Mean	SD		
L/G RATIO AT 0 HRS	0.45	0.22	1.71	2.73	-3.338	0.001
L/G RATIO AT 1 HRS	0.53	0.73	2.56	4.79	-2.959	0.004
L/G RATIO AT 3 HRS	0.57	0.36	3.42	5.40	-3.846	0.001
L/G RATIO AT 6 HRS	0.46	0.29	3.00	3.07	-5.981	0.001
L/G RATIO AT 12 HRS	0.50	0.37	4.97	3.37	-9.566	0.001
L/G RATIO AT 24 HRS	0.41	0.22	2.36	1.76	-7.905	0.001
L/G RATIO AT 48 HRS	0.38	0.35	4.45	6.65	-4.836	0.001

Figure 16: Mean Capillary Lactate/Glucose Ratio at individual intervals for both Groups



In Group A flaps the mean L/G ratio remained stabilized between 0.38 to 0.57 whereas in Group B flaps there was a wide range of L/G ratio varying between 1.71 to 4.97.

Also, in group A flaps the trends in the change of lactate, glucose and L/G ratio is depicted in table 28 & figure 17. There is seen an initial rise in capillary lactate and L/G ratio levels which stabilises in 12-24 hours.

Table 28: Trends in the change in mean of Lactate, Glucose and L/G ratio at 0,1,3,6,12,24,48 hours for group A flaps

Group A	LACTATE	GLUCOSE	L/G RATIO
AT 0 HRS	3.94	8.58	0.45
AT 1 HR	3.81	8.16	0.53
AT 3HRS	4.06	7.59	0.57
AT 6HRS	3.81	7.89	0.46
AT 12 HRS	3.65	7.51	0.50
AT 24 HRS	2.86	6.89	0.41
AT 48 HRS	2.49	6.98	0.38

Figure 17: Trends in the change in mean of Lactate, Glucose and L/G ratio at 0,1,3,6,12,24,48 hours for group A flaps

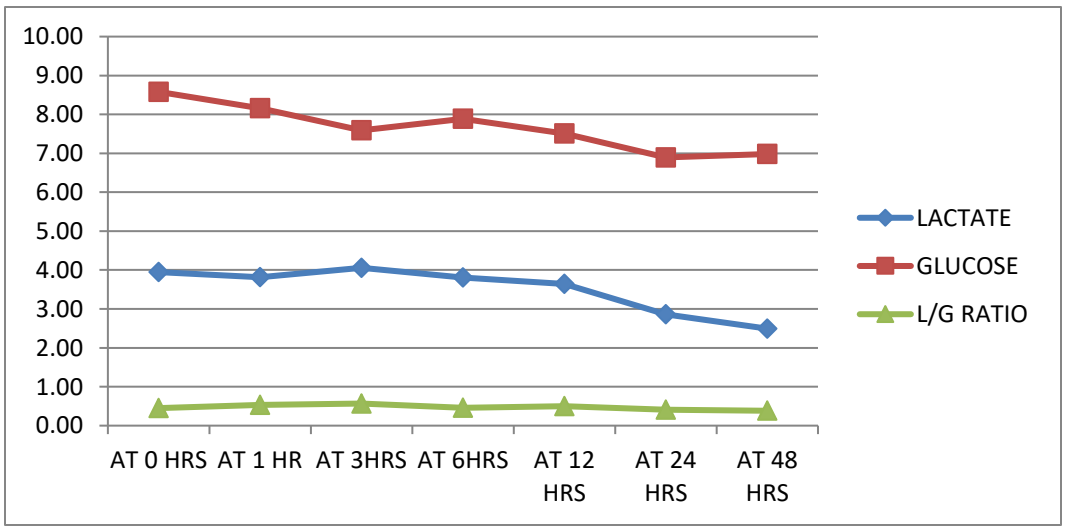
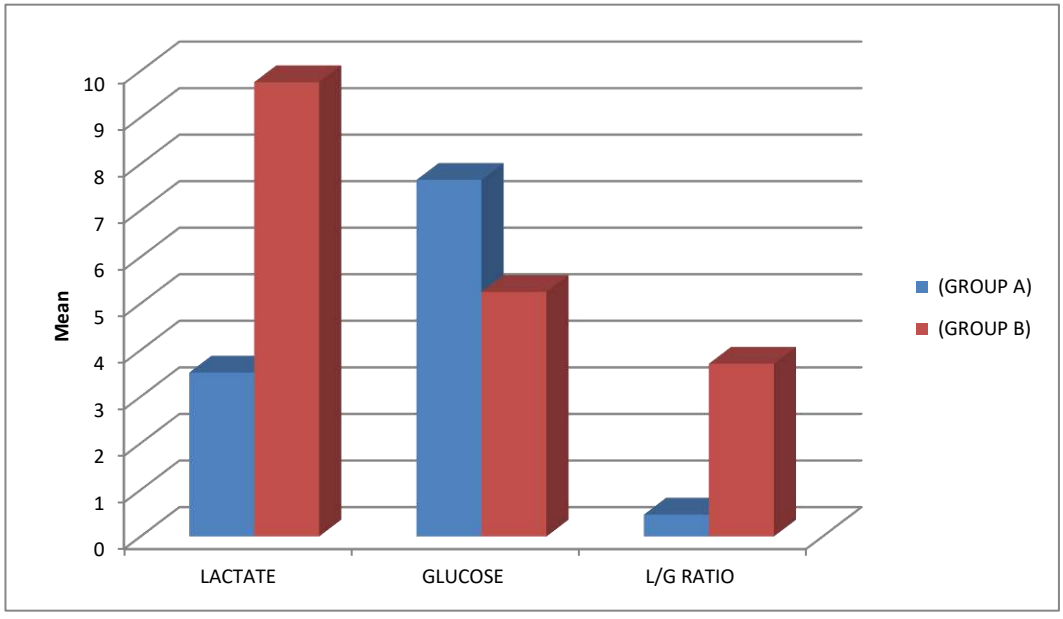


Table 29 & Figure 18 reveals that mean of capillary Lactate and L/G Ratio (LGR) in Group B flaps was more than Group A flaps and that of Capillary Glucose was more in Group A Flaps depicting the increase in shift to anaerobic metabolism in group B.

TABLE 29: Comparison of mean of Lactate, Glucose and L/G ratio in both groups

	Flap without complication (Group A) N=51		**Flap with complication (GROUP B) N=12**		Z	p-value
	Mean	SD	Mean	SD		
LACTATE	3.52	1.56	9.76	3.83	-9.024	0.001
GLUCOSE	7.66	1.37	5.25	1.98	5.009	0.001
L/G RATIO	0.47	0.24	3.71	3.18	-7.378	0.001

Figure 18: Comparison of mean of Lactate, Glucose and L/G ratio in both groups

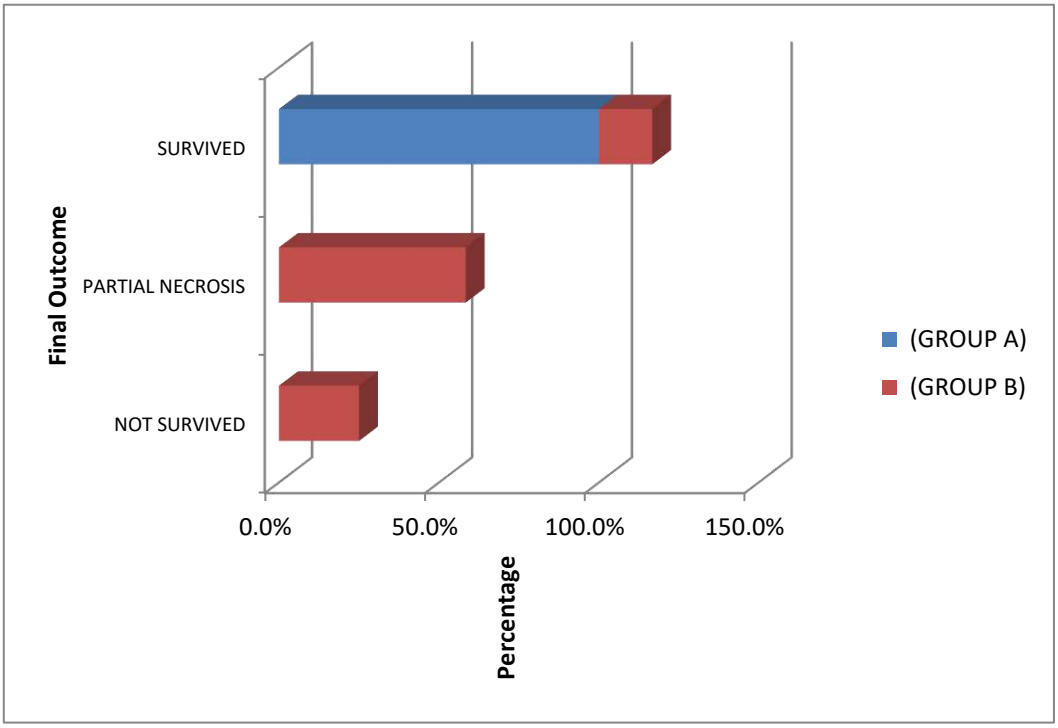


Among the 12 flaps in group B, 3 flaps did not survive at all whereas 7 out of 12 showed signs of partial or marginal necrosis and 2 flaps survived well. (Table 30 & Figure 29)

TABLE 30: Final outcome of flaps in both groups

		Flap without complication (Group A) N=51		**Flap with complication (GROUP B) N=12**		Total	Chi-square value Total	p-value
		No. of cases	(GROUP A)	No. of cases	(GROUP B)			
FINAL RESULTS	NOT SURVIVED	0	0.0%	3	25.0%	3	50.519	0.001
	PARTIAL NECROSIS	0	0.0%	7	58.3%	7		
	SURVIVED	51	100.0%	2	16.7%	53		
Total		51	100.0%	12	100.0%	63		

Figure 19: Final outcome of Flaps in both groups



RECEIVER OPERATOR CURVE ANALYSIS:

ROC analysis was done for Lactate, Glucose and L/G Ratio and the results were as follows:

ROC ANALYSIS FOR CAPILLARY LACTATE (Figure 20 & Table 31, table 32)

Figure 20: ROC for capillary lactate

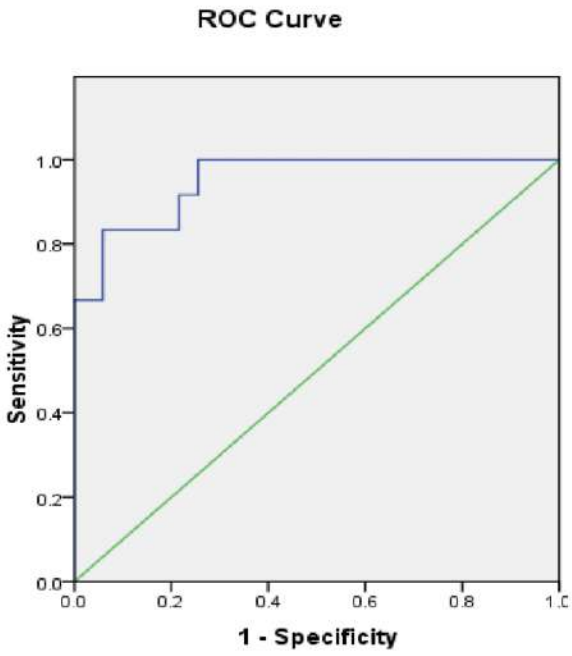


Table 31: Area Under the Curve for Lactate

Area Under the Curve				
Test Result Variable(s): LACTATE				
			Asymptotic 95% Confidence Interval	
Area	Std. Error[a]	p-value	Lower Bound	Upper Bound
.951	.029	.001	.875	1.000

Table 32: Coordinates of the Curve for Lactate

Test Result Variable(s):LACTATE				
Positive if Greater Than or Equal To[a]	Sensitivity	1 - Specificity	Specificity	Youden index
.457	1.000	1.000	0.000	0.000
1.479	1.000	.980	0.020	0.020
1.651	1.000	.961	0.039	0.039
1.907	1.000	.922	0.078	0.078
2.064	1.000	.902	0.098	0.098
2.124	1.000	.882	0.118	0.118
2.181	1.000	.863	0.137	0.137
2.250	1.000	.843	0.157	0.157
2.286	1.000	.824	0.176	0.176
2.314	1.000	.804	0.196	0.196
2.350	1.000	.784	0.216	0.216
2.393	1.000	.765	0.235	0.235
2.429	1.000	.745	0.255	0.255
2.447	1.000	.725	0.275	0.275
2.483	1.000	.706	0.294	0.294
2.521	1.000	.667	0.333	0.333
2.593	1.000	.647	0.353	0.353
2.686	1.000	.627	0.373	0.373
2.729	1.000	.608	0.392	0.392
2.758	1.000	.588	0.412	0.412
2.786	1.000	.569	0.431	0.431
2.857	1.000	.549	0.451	0.451
2.929	1.000	.510	0.490	0.490
2.971	1.000	.490	0.510	0.510
3.050	1.000	.471	0.529	0.529
3.114	1.000	.451	0.549	0.549
3.171	1.000	.431	0.569	0.569
3.350	1.000	.412	0.588	0.588
3.514	1.000	.392	0.608	0.608
3.550	1.000	.373	0.627	0.627
3.650	1.000	.353	0.647	0.647
3.757	1.000	.333	0.667	0.667
3.879	1.000	.314	0.686	0.686
4.093	1.000	.294	0.706	0.706
4.214	1.000	.275	0.725	0.725
4.264	1.000	.255	0.745	0.745
4.314	.917	.255	0.745	0.662

4.371	.917	.235	0.765	0.681
4.457	.917	.216	0.784	0.701
4.564	.833	.216	0.784	0.618
4.814	.833	.196	0.804	0.637
5.064	.833	.176	0.824	0.657
5.150	.833	.157	0.843	0.676
5.386	.833	.137	0.863	0.696
5.621	.833	.118	0.882	0.716
5.886	.833	.098	0.902	0.735
6.179	.833	.078	0.922	0.755
6.539	**.833**	**.059**	**0.941**	**0.775**
6.865	.750	.059	0.941	0.691
6.926	.667	.059	0.941	0.608
7.143	.667	.039	0.961	0.627
7.779	.667	.020	0.980	0.647
8.371	.667	.000	1.000	0.667
8.570	.583	.000	1.000	0.583
9.241	.500	.000	1.000	0.500
10.671	.417	.000	1.000	0.417
11.679	.333	.000	1.000	0.333
12.829	.250	.000	1.000	0.250
14.200	.167	.000	1.000	0.167
15.217	.083	.000	1.000	0.083
16.833	.000	.000	1.000	0.000

ROC ANALYSIS FOR CAPILLARY GLUCOSE (Figure 21 & Table 33, table 34)

Figure 21: ROC for capillary Glucose

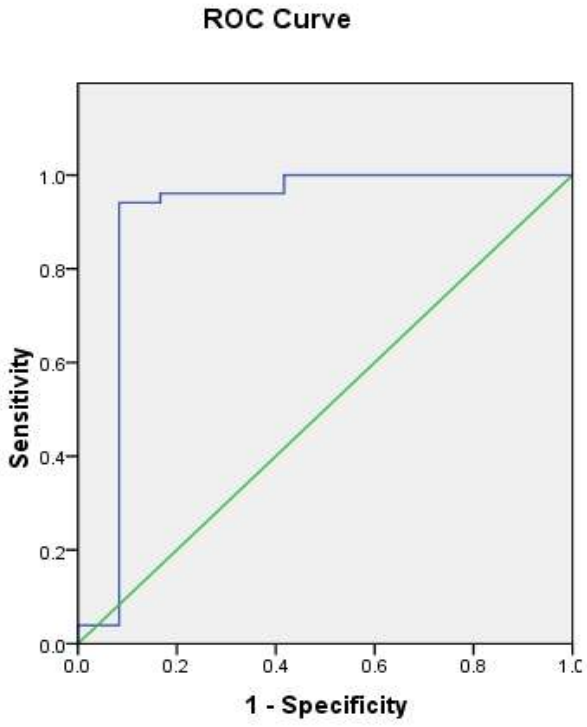


Table 33: Area Under the Curve for Glucose

Area Under the Curve				
Test Result Variable(s): GLUCOSE				
			Asymptotic 95% Confidence Interval	
Area	Std. Error[a]	p-value	Lower Bound	Upper Bound
.905	.076	.001	.000	1.000

Table 34: Coordinates of the Curve for Glucose

Coordinates of the Curve				
Test Result Variable(s):GLUCOSE				
Positive if Greater Than or Equal To[a]	Sensitivity	1 - Specificity	Specificity	Youden index
.980	1.000	1.000	0.000	0.000
2.808	1.000	.917	0.083	0.083
3.988	1.000	.833	0.167	0.167
4.412	1.000	.750	0.250	0.250
4.538	1.000	.667	0.333	0.333
4.816	1.000	.583	0.417	0.417
5.226	1.000	.500	0.500	0.500
5.426	1.000	.417	0.583	0.583
5.483	.980	.417	0.583	0.564
5.562	.961	.417	0.583	0.544
5.603	.961	.333	0.667	0.627
5.694	.961	.250	0.750	0.711
5.899	.961	.167	0.833	0.794
6.095	.941	.167	0.833	0.775
6.201	**.941**	**.083**	**0.917**	**0.858**
6.387	.922	.083	0.917	0.838
6.563	.902	.083	0.917	0.819
6.633	.882	.083	0.917	0.799
6.699	.863	.083	0.917	0.779
6.762	.843	.083	0.917	0.760
6.822	.824	.083	0.917	0.740
6.856	.804	.083	0.917	0.721
6.880	.784	.083	0.917	0.701
6.910	.765	.083	0.917	0.681
6.946	.745	.083	0.917	0.662
6.959	.725	.083	0.917	0.642
6.968	.706	.083	0.917	0.623
6.998	.686	.083	0.917	0.603
7.052	.667	.083	0.917	0.583
7.096	.647	.083	0.917	0.564
7.152	.627	.083	0.917	0.544
7.204	.608	.083	0.917	0.525
7.239	.588	.083	0.917	0.505
7.286	.569	.083	0.917	0.485
7.342	.549	.083	0.917	0.466
7.378	.529	.083	0.917	0.446
7.386	.510	.083	0.917	0.426
7.421	.490	.083	0.917	0.407

7.507	.471	.083	0.917	0.387
7.568	.451	.083	0.917	0.368
7.589	.431	.083	0.917	0.348
7.609	.412	.083	0.917	0.328
7.654	.392	.083	0.917	0.309
7.774	.373	.083	0.917	0.289
7.855	.353	.083	0.917	0.270
7.861	.333	.083	0.917	0.250
7.869	.314	.083	0.917	0.230
7.904	.294	.083	0.917	0.211
7.984	.275	.083	0.917	0.191
8.114	.255	.083	0.917	0.172
8.226	.235	.083	0.917	0.152
8.297	.216	.083	0.917	0.132
8.344	.196	.083	0.917	0.113
8.378	.176	.083	0.917	0.093
8.436	.157	.083	0.917	0.074
8.556	.137	.083	0.917	0.054
8.717	.118	.083	0.917	0.034
8.879	.098	.083	0.917	0.015
9.047	.078	.083	0.917	-0.005
9.436	.059	.083	0.917	-0.025
10.070	.039	.083	0.917	-0.044
10.470	.039	.000	1.000	0.039
12.509	.020	.000	1.000	0.020
15.471	.000	.000	1.000	0.000

ROC ANALYSIS FOR CAPILLARY LACTATE/GLUCOSE RATIO: (Figure 22 & Table 35, Table 36)

Figure 22: ROC for capillary L/G ratio

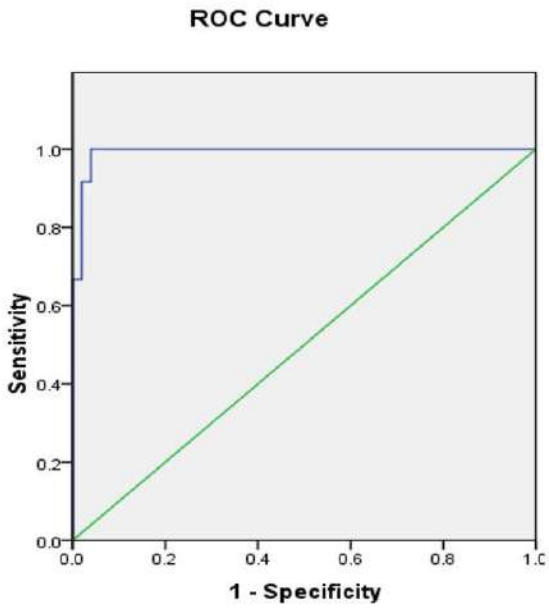


Table 35: Area Under the Curve for L/G ratio

Area Under the Curve				
Test Result Variable(s): L/G RATIO				
Area	Std. Error[a]	p-value	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.992	.008	.001	.000	1.000

Table 36: Coordinates of the Curve for L/G ratio

Coordinates of the Curve				
Test Result Variable(s):L/G RATIO				
Positive if Greater Than or Equal To[a]	Sensitivity	1 - Specificity	Specificity	Youden index
-.809	1.000	1.000	0.000	0.000
.194	1.000	.980	0.020	0.020
.232	1.000	.961	0.039	0.039
.280	1.000	.922	0.078	0.078
.294	1.000	.902	0.098	0.098
.299	1.000	.882	0.118	0.118
.306	1.000	.863	0.137	0.137
.310	1.000	.843	0.157	0.157
.314	1.000	.824	0.176	0.176
.316	1.000	.804	0.196	0.196
.321	1.000	.784	0.216	0.216
.327	1.000	.765	0.235	0.235
.331	1.000	.745	0.255	0.255
.333	1.000	.725	0.275	0.275
.334	1.000	.706	0.294	0.294
.337	1.000	.686	0.314	0.314
.344	1.000	.667	0.333	0.333
.349	1.000	.647	0.353	0.353
.351	1.000	.627	0.373	0.373
.358	1.000	.608	0.392	0.392
.364	1.000	.588	0.412	0.412
.368	1.000	.569	0.431	0.431
.374	1.000	.549	0.451	0.451
.380	1.000	.510	0.490	0.490
.385	1.000	.490	0.510	0.510
.399	1.000	.471	0.529	0.529
.417	1.000	.451	0.549	0.549
.425	1.000	.431	0.569	0.569
.442	1.000	.412	0.588	0.588
.460	1.000	.392	0.608	0.608
.464	1.000	.373	0.627	0.627
.477	1.000	.353	0.647	0.647
.495	1.000	.333	0.667	0.667
.513	1.000	.314	0.686	0.686
.541	1.000	.294	0.706	0.706
.568	1.000	.275	0.725	0.725
.578	1.000	.255	0.745	0.745

.581	1.000	.235	0.765	0.765
.584	1.000	.216	0.784	0.784
.586	1.000	.196	0.804	0.804
.597	1.000	.176	0.824	0.824
.611	1.000	.157	0.843	0.843
.631	1.000	.137	0.863	0.863
.659	1.000	.118	0.882	0.882
.701	1.000	.098	0.902	0.902
.768	1.000	.078	0.922	0.922
.862	1.000	.059	0.941	0.941
.988	**1.000**	**.039**	**0.961**	**0.961**
1.112	.917	.039	0.961	0.877
1.207	.917	.020	0.980	0.897
1.257	.833	.020	0.980	0.814
1.296	.750	.020	0.980	0.730
1.422	.667	.020	0.980	0.647
1.802	.667	.000	1.000	0.667
2.504	.583	.000	1.000	0.583
3.060	.500	.000	1.000	0.500
3.329	.417	.000	1.000	0.417
3.463	.333	.000	1.000	0.333
3.834	.250	.000	1.000	0.250
6.976	.167	.000	1.000	0.167
10.118	.083	.000	1.000	0.083
11.487	.000	.000	1.000	0.000

On analysing the ROC curve for Capillary lactate, capillary Glucose and L/G Ratio, all 3 were found to be good parameters for flap monitoring with L/G Ratio having maximum Area under the curve making L/G Ratio (LGR) a best predictor among the 3.

On detail analysing the co-ordinates of the ROC curve and calculating the Youden Index, we were able to predict the cut off values of each parameter as highlighted in the charts above and summarised in table 37 & Figure 23:

Cut off value of lactate = 6.5 at 83% sensitivity and 94% specificity with AUC 0.951

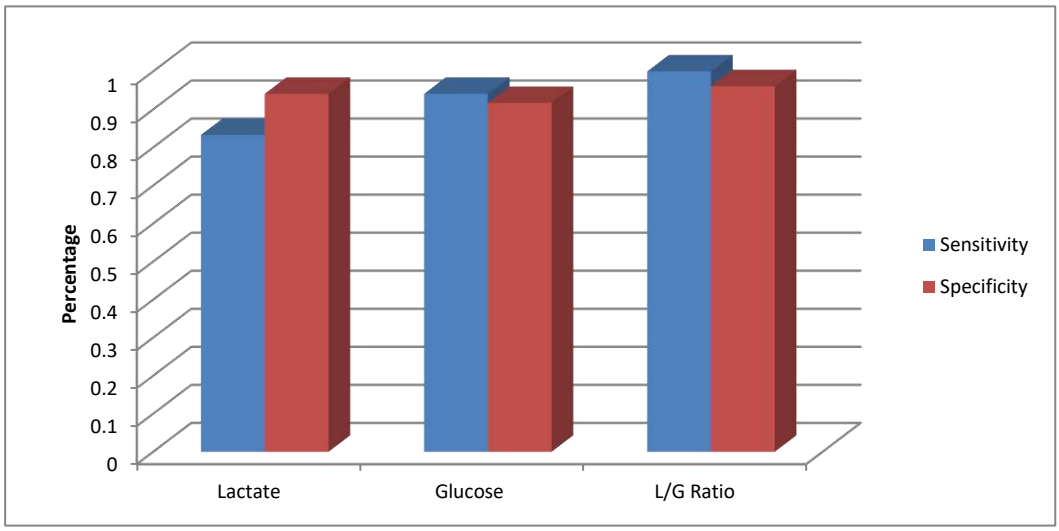
Cut off value of glucose = 6.2 at 94% sensitivity and 91.7% specificity with AUC O.905

Cut off value of L/G = 0.98 (1) at 100% sensitivity and 96% specificity with AUC 0.992

TABLE 37: Cut off Values as per ROC curve analysis

	Cut off value	**Sensitivity**	**Specificity**	**Youden index**
Lactate	6.539	0.833	0.941	0.775
Glucose	6.201	0.941	0.917	0.858
L/G Ratio	.988	1	0.961	0.961

Figure 23: Cut off Values as per ROC curve analysis



Taking 0.98 as the cut off value of L/G ratio, it was found that clinical signs of complications appeared later than that predicted by L/G Ratio. The rise in LGR occurred approximately 1 – 48 hours before the appearance of clinical signs as shown in Table no. 38.

TABLE 38: Appearance of clinical signs relative to the rise in L/G ratio (LGR) above cut off

FLAP NAME	FLAP TYPE	Appearance of clinical signs	LGR above the cut off >1	Difference
ALT FREE FLAP	FREE	1 hour	0 hour	1 hour
GROIN FLAP	PEDICLED	12 hour	6 hour	6 hour
ALT FREE FLAP	FREE	12 hour	1 hour	11 hour
PROPELLER FLAP	PEDICLED	24 hour	1 hour	23Hour
FASCIOCUTANEOUS TRANSPOSITION FLAP	PEDICLED	12 hour	0 hour	12Hour
ALT FREE FLAP	FREE	12 hour	1 hour	11Hours
PROPELLER FLAP	PEDICLED	12 hour	3 hour	9Hour
CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	48 hour	0 hour	48Hour
REVERSE SURAL ARTERY FLAP	PEDICLED	6 hour	0 hour	6Hour
CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	24 hour	6 hour	18Hour
CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	48 hour	12 hour	24Hour
ALT FREE FLAP	FREE	24 hour	0 hour	24Hour

The ROC curve of LGR was found comparative to that of Lactate As shown in Figure 24 below. So, when this cut off value is used to predict the survival of the flaps in both the groups, False Positives and False Negatives are identified as in Table 39 and Table 40.

Figure 24: Comparison of ROC of LGR and Lactate

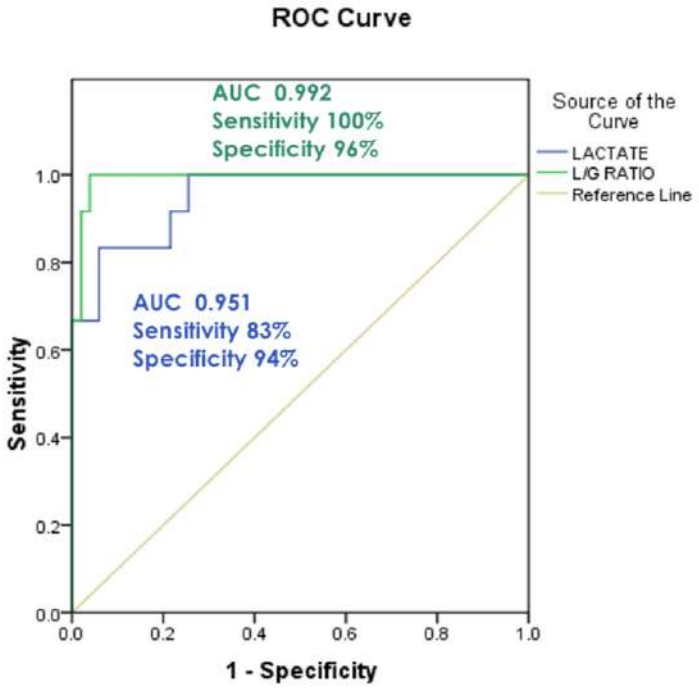


Table 39 Indicates 13 flaps had lactate value above its cut off among which 3 (23.1%) belonged to Group A (False Positives). And among 50 flaps that had lactate value below its cut off, 2 flaps belonged to group B (False Negatives)

TABLE 39: Frequency distribution of group A and group B flaps according to cut off value of lactate

		Flap without complication (Group A) N=51		**Flap with complication (Group B) N=12**		Total	Chi-square valueTotal	p-value
		No. of cases	Percentage	No. of cases	Percentage			
Lactate	< 6.54	48(True Positive)	96.0%	**2 (False Negative)**	4.0%	50	35.582	0.001
	> 6.54	**3 (False Positive)**	23.1%	10(True Negative)	76.9%	13		
Total	51		81.0%	12	19.0%	63		

Similarly, among 14 flaps that had LGR above its cut off, 2 flaps (14.3%) belonged to Group A (False Positives). And among 49 flaps that had lactate value below its cut off, none belonged to group B (False Negatives) as depicted in table 40.

TABLE 40: Frequency distribution of group A and group B flaps according to cut off value of L/G ratio (LGR)

		Flap without complication (Group A) N=51		**Flap with complication (Group B) N=12**		Total	Chi-square value Total	p-value
		No. of cases	Percentage	No. of cases	Percentage			
L/G RATIO	< 0.98	49 (True Positive)	100.0%	**0 (False Negative)**	0.0%	49	51.882	0.001
	> 0.98	**2 (False Positive)**	14.3%	12 (True Negative)	85.7%	14		
Total		51	81.0%	12	19.0%	63		

DISCUSSION

Flap survival is critical in the outcome of any reconstructive surgery and the ability to detect flap complications early and accurately is important in improving the results. We and many authors believe that Accuracy, Efficiency and Objectivity are the keys to successful flap monitoring.[13]

Lesser is the time between the onset of vascular compromise and salvage interventions to correct them, more are the chances of flap survival therefore necessitating a reliable monitoring tool.[46]

Though clinical monitoring of flaps is still considered as “Gold Standard” method of assessment of flap viability [47] but on revisiting the literature, we understand that the clinical signs may take long time to appear following the pedicle impairment making its salvage difficult and challenging.[3] Also, clinical monitoring alone is not always adequate to detect vascular compromise. [48]

We know all cells metabolise aerobically in the presence of oxygen which is derived from blood and in absence of oxygen because of decreased blood supply, cells enter anaerobic respiration whose end product is lactate. In cases of flap monitoring, capillary glucose and lactate values are calculated from the tissue and their ratio (Lactate/Glucose) is said to be significant in assessment of metabolic status of the tissue. An increased Lactate/Glucose Ratio (LGR) indicates a shift towards anaerobic metabolism, which predicts compromised vascularity of flap.

Based on this physiology of cell metabolism, several objective methods of flap monitoring are being practiced among which microdialysis has gained much importance.[15,16] Microdialysis continuously measures glucose, lactate, pyruvate using an in dwelling catheter. But because of its cost, invasiveness and the need for surgical expertise, its use is limited.

Various studies have suggested the use of simpler, non-invasive and cheaper alternatives than microdialysis for measurement of markers like Capillary Lactate and Capillary Glucose for predicting flap survival but

there is scarce data available showing the role of Capillary Lactate to Glucose Ratio (LGR) in predicting flap survival.

This present study was conducted to assess the use of lactate/glucose ratio (LGR) as a potential predictor of flap viability in reconstructive surgery.

Over a period of 17 months, from August 2021 to December 2022, a total of 63 flaps (both Free and pedicled) were studied.

All the diabetic and critically ill patients were excluded from the study as their existing systemic metabolic derangements might affect the values of glucose and lactate.

On studying the patient characteristics, the minimum age was 13 years, and the maximum age was 74 years with mean of 42±14.5 years. Majority of these patients (49.2%) belong to the middle age group (31-50 years). Male preponderance was seen (88.9%).

Head and neck cancer was the most common cause of defect followed by Trauma, thus making head and neck as most common site requiring coverage.

Venous insufficiency was more common (58.3%) cause of flap complications than arterial insufficiency as also mentioned by other authors.[3-8]

In present study, clinical monitoring of flap included 4 parameters-color, temperature, capillary refill and bleeding characteristics. These observations were done every hour and were recorded at particular designated intervals (0,1,3,6,12,24 and 48 hours) along with capillary Lactate and Glucose levels (from the distal 1/3rd of the flap) and the values in mmol/L were then used to calculate the LGR. The decision to intervene surgically was taken solely on the basis of clinical assessment based on surgeon’s experience.

It was observed that by the end of 48 hours, a total of 12 flaps showed complications. All these flaps were included in Group B whereas

all the remaining healthy flaps that did not develop complications were included in Group A.

Analysis of association of outcome of flap on the basis of clinical evaluation was done by chi-square test and the results implicated statistically significant association at and after 6 hours with P value <0.005.

On comparing both groups in terms of Mean Lactate, glucose and LGR at all intervals, a significant correlation was observed ($p<0.005$) with exception of Glucose at 24 and 48 hours where the association was non-significant statistically.

The Mean values of Lactate in Group A and Group B were 3.52 ± 1.56 mmol/L and 9.76 ± 3.83 mmol/L respectively. The mean values for Glucose was 7.66 ± 1.37 mmol/L and 5.25 ± 1.98 mmol/L respectively. The calculated mean of LGR it was 0.47 ± 0.24 and 3.71 ± 3.18 respectively. As also observed by Sakakibara et al., the mean Glucose value in our study was lower in Group B flaps.[29] The mean Lactate and LGR was persistently higher in Group B flaps as observed by Setala et al. in 2013.[38]

The cut off value of Lactate, Glucose and LGR was calculated by using ROC analysis.

Though various studies observed cut off for glucose as 62mg/dl, but in present study, the cut off came out to be 6.2mmol/L equivalent to 52mg/dl at sensitivity and specificity of 94.1% and 91.7% respectively.[28,32,33]

We attained a Cut off value of 6.5 for lactate at sensitivity of 83% and specificity of 94% which was comparable to that of 6.4 in studies conducted by Henault et al at sensitivity of 100% and specificity of 96.9%.[43] Jyranki et al., in a clinical study, reported the cut off value of 7mmol/L below which the flaps were without impeded blood flow.[17]Also, according to Bakker et al., the normal value of lactate is 2mmol/L and the raised mean value of 6.6mmol/L can be seen in pathological conditions or

multi organ failure.[49] Kindermann et al. reported that at blood lactate value >4.0mmol/L, the speed of production of lactate was more than the speed at which it is metabolised, leading it to accumulate.[50]

In our study, the change in Lactate was more sensitive than the change in glucose for group B flaps. This finding of ours coincides with that of Keita Kishi et al.[45]

Our cut off value for LGR was 0.98 at 100% sensitivity and 96% specificity which was comparable to that predicted by Chi-Tsung Su et al.[49]and Setala et al in their study.[38] Sorensen reported the similar sensitivity and specificity in his experimental study.[35]

In our study, the Area under Receiver operator curve (AUROC) of Lactate (0.951) and LGR (0.992) were higher than that of Glucose (0.905) and on comparing both we observed that if this cut off value of LGR was considered during the flap monitoring, 2 otherwise healthy flaps may have undergone intervention (False-Positives). On the other hand, the flaps of Group B which did not survive could have been salvaged by early and timely intervention.

According to a study, such False Positive values especially in initial hours may be attributed to the delay in vascularisation as in case of free flaps.[31] Hence omission of much initial measurements is advised in study conducted by Henault et al.[43] Contrary to that, it is not true in our case as in 5 out of 12 cases of Group B, the cut off threshold was observed at 0 hour itself which cannot be ignored.

Also, if only a single marker- Lactate was taken for monitoring not only the false positive rate, but the False Negative rate was also higher as compared to taking LGR alone for assessment. Thus, LGR is a more accurate and better marker than Lactate or glucose monitoring alone as is also mentioned by Hara et al.[28]

According to Setala et al, and in experimental study by Sorensen et al., sudden rise in L/G Ratio to hundreds is a strong indicator of ischaemia but in our study we did not observe any value in hundreds.[35,38]

Many previous studies used microdialysis to measure flap glucose and lactate.[35,37] Microdialysis uses an embedded catheter which allows continuous monitoring and has excellent objectivity but the complicated procedure and need for professional expertise has been cited as disadvantages.[43] By contrast, many studies support the use of simple measuring instruments to measure glucose and lactate.[29,43] However, the major drawback of this easy and cost effective method is its inability to access the buried flaps or inability to monitor glucose and lactate values of pure muscle flaps which can be easily evaluated by the use of implantable catheter of microdialysis.

Setala et al.[35] were also able to differentiate between arterial and venous thrombosis as the cause of flap failure by the use of Microdialysis as reported by few other studies but in our case, this differentiation was not possible. This result of our study is supported by Henault et al. who also used glucometer and lactometer and believed this differentiation non-significant as surgical intervention of impaired pedicles include exploration of both artery as well as vein. [43,52,53]

Moreover, application of this tool in our study has revealed that LGR rises well before the appearance of clinical signs of flap compromise. Similar finding was observed in many studies.[36,38,43] According to Henault et al., the threshold-couple or the cut-off was reached approximately 5.4 hours before the appearance of clinical signs of pedicle impairment.[43] Setala et al. reported that lactate rises 1-12 hours before the clinical signs emerged and the glucose values decreased 2-8 hours before the clinical findings.[37]But which value was first to react was not clarified in our study.

Since we used the cut off values retrospectively to findout an objective, reliable and best screening tool, the salvage rate was not affected.

This also proved to be a cost-effective tool as compared to microdialysis which is expensive and invasive. The Lactate pro device is similar to Glucometer. It utilizes disposable strips and also needs

calibration with new strip set. And the total cost of consumables for 48 hours of flap monitoring cost less than 2500 INR.

The role of LGR marker cannot be established in Diabetic patients in whom the flap capillary glucose levels may get affected by the systemic hypo or hyperglycemia and indirectly altering the LGR. In these patients, capillary Lactate values alone can be reliable indicator as the lactate rate is least affected by the systemic glucose levels. Also, metabolic abnormalities in cancer patients can cause lactic acid impairment in solid tumors and hepatic dysfunction in chronic alcoholic patients, all can alter the lactate levels but in present study these issues were not assessed and we excluded diabetic and critically ill patients. To prove or disprove this, more studies on LGR in selective patients need to be conducted.

Our study utilized lactometer (Lactate Scout 4) which requires only 0.2 microlitre of capillary blood[54] which is much less than the blood drop size required by the Glucometer (Freestyle Optium Neo) that needs 0.6 microlitres of capillary blood.[55] The smaller size of drop is an advantage of using lactate alone as a marker over glucose or LGR in flaps where a good size drop cannot be formed on pin prick. Such cases were excluded from our study.

The number of pin pricks can no doubt cause cosmetically unappealing results temporarily like scarring, but the patients and the surgeons are definitely more concerned about the flap survival and salvage.

Overall, our results predict that LGR is a more sensitive screening tool as compared to Glucose or Lactate. It can be safely used as an adjunct to the Gold Standard. Its objectivity can help save manpower and even a nursing staff in the Recovery Room can easily perform this test at dedicated intervals and inform the surgeon when the cut off value is approached making the surgeon more vigilant. This simple measuring method is also more reliable in case of intra oral flap monitoring where

clinical evaluation cannot be relied upon as it is hindered by unfavorable environmental conditions like insufficient light.

At the same time, to reinforce this as an efficient, objective screening test, similar studies on large number of flaps need to be conducted taking the cut off value as the deciding factor for Redo surgeries.

SUMMARY & CONCLUSION

A prospective study was conducted in the department of Plastic and Reconstructive Surgery at Dayanand Medical College and Hospital to study the role of capillary Lactate/Glucose Ratio in Flap monitoring. All the patients undergoing any kind of flap surgery from August 2021 to December 2022 and meeting the inclusion criteria were included in the study. All diabetic and critically ill patients were excluded from the study.

Clinical monitoring of flap was done and findings were recorded at 0,1,3,6,12,24 and 48 hours along with flap capillary lactate and glucose values (from distal 1/3rd margin of flap) in mmol/L. The lactate and glucose values were utilised to calculate the LGR (L/G Ratio). The decision to intervene surgically was taken solely on the basis of clinical assessment based on surgeon's experience.

It was observed that by the end of 48 hours, a total of 12 flaps showed complications. All these flaps were included in Group B whereas all the remaining healthy flaps that did not develop complications were included in Group A.

On the basis of our statistical analysis, we conclude that:

- The use of LGR as a predictor for flap survival is a useful adjunct to clinical monitoring especially in non-diabetics.
- Its high sensitivity makes it an effective screening tool.
- It is cost effective as compared to other tools, is non-invasive, needs no expertise and is objective.
- The cut off value of 0.98 (at 100% sensitivity and 96% specificity) can be considered for flap monitoring above which vascular compromise can be suspected making surgeons more vigilant.
- And since these alarming thresholds are reached well before the appearance of clinical signs, it can aid decision making for redo surgeries and help achieving high flap salvage rate specifically in microsurgery.

- Our study highlights need for a pilot study for early exploration at low LGR needs to be conducted to establish this as an efficient objective monitoring tool for flap viability.

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Picture 3: Radial forearm free flap for face defect

Picture 4: Fasciocutaneous transposition flap for ankle defect

Flap developed marginal discoloration

Sutures removed as salvage intervention

Picture 5: Abdominal flap for forearm defect

Picture 6: PMMC flap for oral cavity and chin

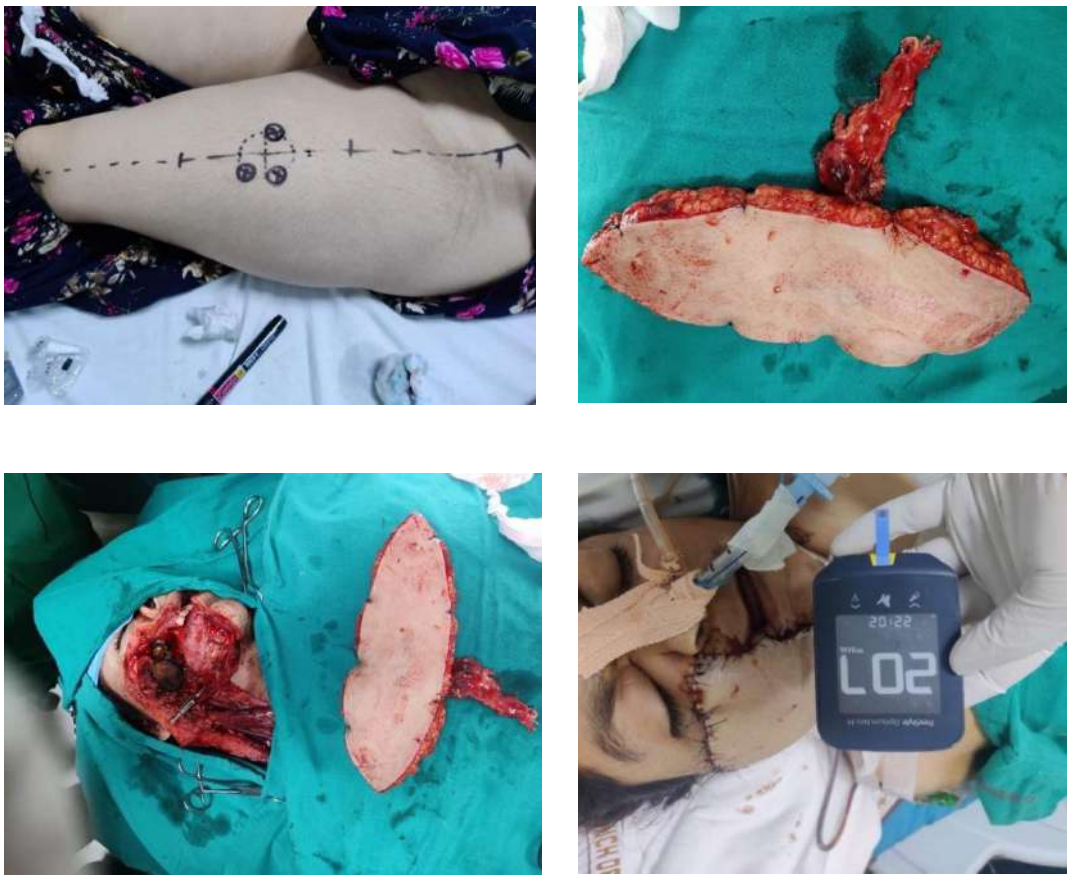
Picture 7: LD free flap for dorsum of foot defect

Picture 8: Propeller flap for ankle defect

Showed venous bleed on pin prick, sutures were removed distally as salvage intervention

Picture 9: Free Fibula Osseocutaneous Flap for mandibular defect

Picture 10: ALT Free flap for face defect



Picture 11: Cross leg flap for exposed tibia

Picture 12: TFL Fasciocutaneous flap for trochanter defect

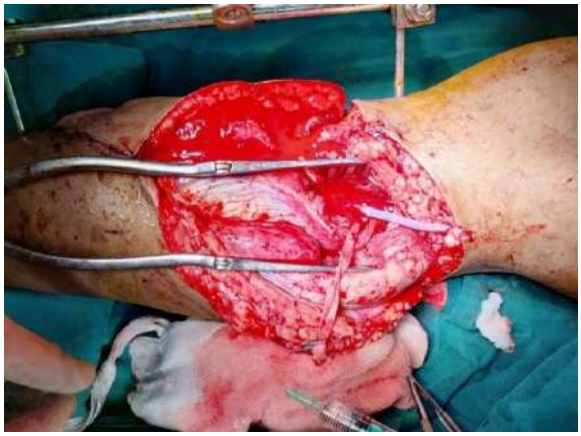
Picture 13: Groin flap for wrist

Picture 14: Gluteal Rotation advancement flap for sacral pressure sore

Picture 15: RSA flap for heel defect

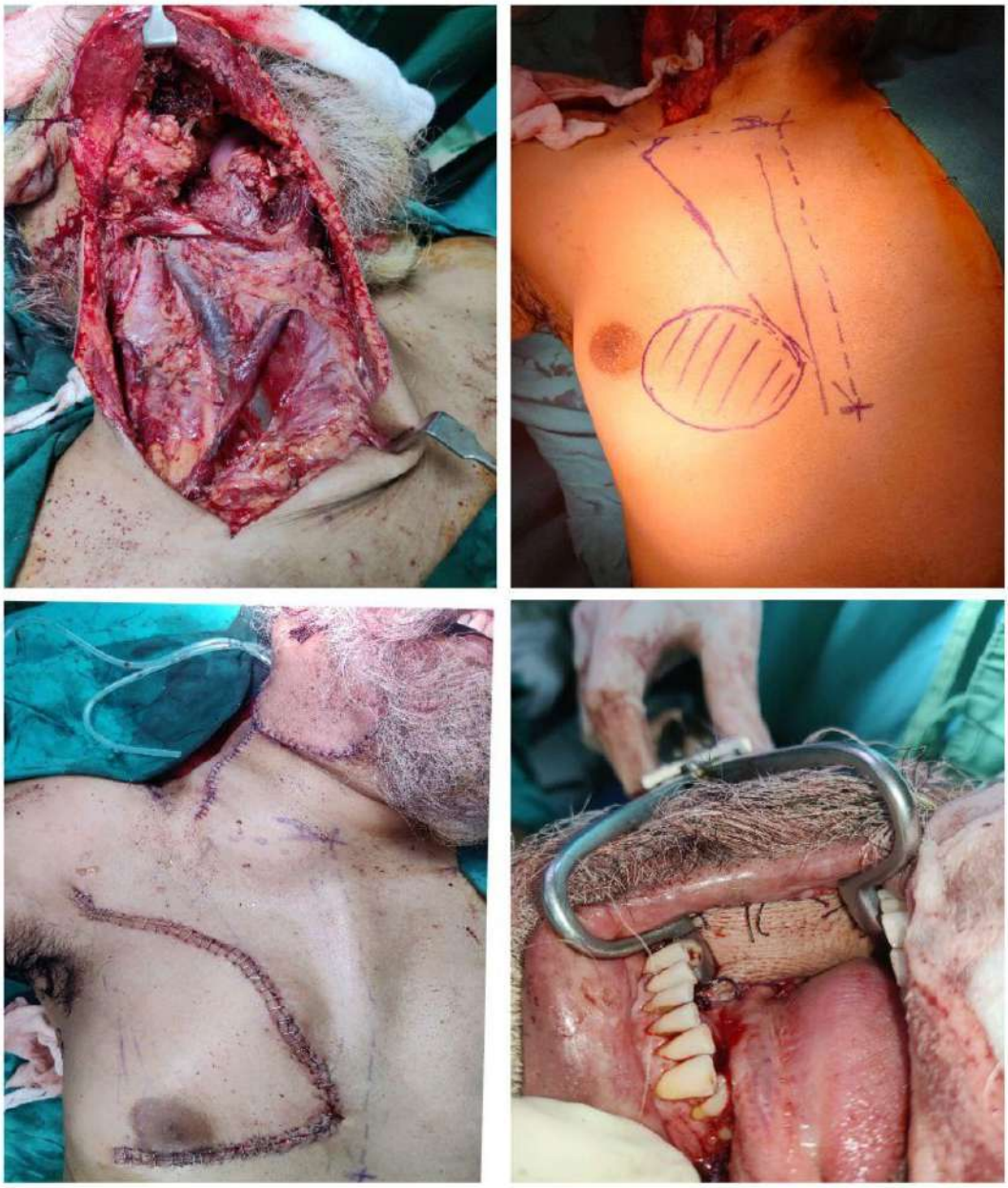
Picture 16: RSA flap for Ankle defect

Picture 17: ALT Free flap for leg defect



Picture 18: Pedicled LD flap for Breast Reconstruction

Picture 19: PMMC flap for intra oral defect



Picture 20: Groin flap for dorsum of hand defect

Picture 21: Radial artery forearm free flap for face defect

S.NO	MRD.NO	NAME	Age	SEX (M/F)	CAUSE OF DEFECT	WOUND SITE	FLAP NAME	FLAP TYPE	COLOUR 0 HRS	COLOUR 1 HRS	COLOUR 3 HRS	COLOUR 6 HRS	COLOUR 12 HRS
1	1130100	SS	17	M	POST TRAUMATIC	LEG	FASCIOCUTANEOUS TRANSPOSITION FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
2	800878	VKV	64	M	PRESSURE SORE	TROCHANTER	TFL MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
3	1130100	SS	17	M	POST TRAUMATIC	ANKLE/FOOT	ALT FREE FLAP	FREE	SAS	BLUISH	BLUISH	SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
4	646015	PK	48	M	CARCINOMA	ANKLE/FOOT	REVERSE SURAL ARTERY FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
5	1121162	MK	29	F	EXTRAVASATION INJURY	FOREARM	GROIN FLAP	PEDICLED	SAS	SAS	SAS	SAS	BLUISH
6	1171751	RS	49	M	HEAD & NECK CANCER	ORAL CAVITY	FREE FIBULA OSTEOCUTANEOUS FLAP	FREE	SAS	SAS	SAS	SAS	SAS
7	1174116	DS	74	M	HEAD & NECK CANCER	ORAL CAVITY	PECTORALIS MAJOR MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
8	1174808	SB	13	F	POST ELECTRIC BURN	HAND	ALT FREE FLAP	FREE	SAS	SAS	SAS	BLUISH	BLUISH
9	1181144	BS	50	M	HEAD & NECK CANCER	ORAL CAVITY	SUBMENTAL FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
10	1201101	SM	14	M	POST TRAUMATIC	LEG	CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
11	1158979	RK	20	M	BULLET INJURY	FACE	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
12	1208033	LS	40	M	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
13	1173637	R	24	M	POST TRAUMATIC	FOREARM	ABDOMINAL FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
14	1218930	SM	44	M	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
15	1219875	SS	70	M	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
16	1221149	MS	50	M	HEAD & NECK CANCER	ORAL CAVITY	FREE FIBULA OSTEOCUTANEOUS FLAP	FREE	SAS	SAS	SAS	SAS	SAS
17	1216461	SS	55	M	POST TRAUMATIC	LEG	FASCIOCUTANEOUS TRANSPOSITION FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
18	1210101	SS	54	M	POST TRAUMATIC	LEG	PROPELLER FLAP	PEDICLED	SAS	SAS	SAS	BLUISH	BLUISH
19	1227316	VK	47	M	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
20	1230796	RS	36	M	POST TRAUMATIC	LEG	FASCIOCUTANEOUS TRANSPOSITION FLAP	PEDICLED	SAS	SAS	SAS	BLUISH	BLUISH
21	1235230	SK	51	M	HEAD & NECK CANCER	ORAL CAVITY	LATISSIMUS DORSI MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
22	1235230	SK	51	M	HEAD & NECK CANCER	ORAL CAVITY	PECTORALIS MAJOR MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
23	1171251	KK	60	M	PRESSURE SORE	SACRUM	GLUTEAL CUTANEOUS ROTATIONAL FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
24	1222115	R	25	M	PRESSURE SORE	SACRUM	GLUTEAL CUTANEOUS ROTATIONAL FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
25	1242471	CS	45	M	HEAD & NECK CANCER	ORAL CAVITY	FREE FIBULA OSTEOCUTANEOUS FLAP	FREE	SAS	SAS	SAS	SAS	SAS
26	1242471	CS	45	M	HEAD & NECK CANCER	ORAL CAVITY	PECTORALIS MAJOR MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
27	1230347	JR	65	M	POST TRAUMATIC	LEG	FASCIOCUTANEOUS TRANSPOSITION FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
28	1232827	RS	52	M	POST TRAUMATIC	FOREARM	ABDOMINAL FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
29	1240051	SS	49	M	POST TRAUMATIC	ANKLE/FOOT	LAT DORSI MYOCUTANEOUS FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
30	1246646	JS	35	M	POST TRAUMATIC	LEG	ALT FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
31	1018151	GS	42	M	HEAD & NECK CANCER	ORAL CAVITY	FREE FIBULA OSTEOCUTANEOUS FLAP	FREE	SAS	SAS	SAS	SAS	SAS
32	1249532	JS	35	M	POST TRAUMATIC	LEG	CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
33	1250327	AS	18	M	POST TRAUMATIC	ANKLE/FOOT	PROPELLER FLAP	PEDICLED	SAS	SAS	SAS	SAS	NOT SURVIVED/MARGINAL NECROSIS
34	1227561	SD	33	M	POST TRAUMATIC	FOREARM	GROIN FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS

S.NO	MRD.NO	COLOUR 24 HRS	COLOUR 48HRS	CAPILLARY REFILL 0 HRS	CAPILLARY REFILL 1 HRS	CAPILLARY REFILL 3 HRS	CAPILLARY REFILL 6 HRS	CAPILLARY REFILL 12 HRS	CAPILLARY REFILL 24 HRS
1	1130100	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
2	800878	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
3	1130100	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	SPONTANEOUS	SPONTANEOUS	DELAYED	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	SURVIVED/MARGINAL NECROSIS
4	646015	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
5	1121162	BLUISH	BLUISH	WNL	WNL	WNL	WNL	SPONTANEOUS	WNL
6	1171751	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
7	1174116	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
8	1174808	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	WNL	WNL	WNL	DELAYED	ABSENT	NOT SURVIVED/MARGINAL NECROSIS
9	1181144	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
10	1201101	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
11	1158979	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
12	1208033	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
13	1173637	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
14	1218930	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
15	1219875	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
16	1221149	SAS	SAS	SPONTANEOUS	SPONTANEOUS	WNL	WNL	WNL	WNL
17	1216461	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
18	1210101	BLUISH	BLUISH	WNL	WNL	DELAYED	DELAYED	DELAYED	DELAYED
19	1227316	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
20	1230796	BLUISH	BLUISH	WNL	WNL	WNL	WNL	WNL	DELAYED
21	1235230	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
22	1235230	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
23	1171251	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
24	1222115	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
25	1242471	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
26	1242471	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
27	1230347	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
28	1232827	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
29	1240051	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
30	1246646	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	WNL	WNL	WNL	WNL	WNL	NOT SURVIVED/MARGINAL NECROSIS
31	1018151	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
32	1249532	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
33	1250327	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	WNL	WNL	WNL	SPONTANEOUS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
34	1227561	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL

S.NO	MRD.NO	CAPILLARY REFILL 48 HRS	BLEEDING CHARACTERISTICS AT 0 HRS	BLEEDING CHARACTERISTICS AT 1 HRS	BLEEDING CHARACTERISTICS AT 3 HRS	BLEEDING CHARACTERISTICS AT 6 HRS	BLEEDING CHARACTERISTICS AT 12 HRS	BLEEDING CHARACTERISTICS AT 24 HRS	BLEEDING CHARACTERISTICS AT 48 HRS
1	1130100	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
2	800878	WNL	ARTERIAL	ARTERIAL	VENOUS	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
3	1130100	NOT SURVIVED/MARGINAL NECROSIS	ARTERIAL	VENOUS	NO BLEED	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
4	646015	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
5	1121162	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	VENOUS	VENOUS	VENOUS
6	1171751	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
7	1174116	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
8	1174808	NOT SURVIVED/MARGINAL NECROSIS	ARTERIAL	VENOUS	VENOUS	VENOUS	VENOUS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
9	1181144	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
10	1201101	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
11	1158979	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
12	1208033	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
13	1173637	WNL	VENOUS	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
14	1218930	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
15	1219875	WNL	ARTERIAL	ARTERIAL	ARTERIAL	VENOUS	VENOUS	ARTERIAL	ARTERIAL
16	1221149	WNL	ARTERIAL	ARTERIAL	VENOUS	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
17	1216461	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
18	1210101	DELAYED	ARTERIAL	ARTERIAL	ARTERIAL	VENOUS	VENOUS	VENOUS	VENOUS
19	1227316	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
20	1230796	DELAYED	ARTERIAL	ARTERIAL	VENOUS	VENOUS	VENOUS	VENOUS	VENOUS
21	1235230	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
22	1235230	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
23	1171251	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
24	1222115	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
25	1242471	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
26	1242471	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
27	1230347	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
28	1232827	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
29	1240051	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
30	1246646	NOT SURVIVED/MARGINAL NECROSIS	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
31	1018151	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
32	1249532	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
33	1250327	NOT SURVIVED/MARGINAL NECROSIS	ARTERIAL	ARTERIAL	ARTERIAL	VENOUS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
34	1227561	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL

S.NO	MRD.NO	TEMPERATURE AT 0 HRS	TEMPERATURE AT 1 HRS	TEMPERATURE AT 3 HRS	TEMPERATURE AT 6 HRS	TEMPERATURE AT 12 HRS	TEMPERATURE AT 24 HRS	TEMPERATURE AT 48 HRS	OUTCOME OF FLAP AT 0 HRS	OUTCOME OF FLAP AT 1 HRS	OUTCOME OF FLAP AT 3 HRS
1	1130100	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
2	800878	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
3	1130100	SAS	COLD	COLD	SURVIVED/MARGINAL NECROSIS	SURVIVED/MARGINAL NECROSIS	SURVIVED/MARGINAL NECROSIS	SURVIVED/MARGINAL NECROSIS	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS
4	646015	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
5	1121162	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
6	1171751	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
7	1174116	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
8	1174808	SAS	SAS	SAS	SAS	COLD	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	SURVIVED	SURVIVED	SURVIVED
9	1181144	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
10	1201101	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
11	1158979	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
12	1208033	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
13	1173637	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
14	1218930	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
15	1219875	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
16	1221149	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
17	1216461	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
18	1210101	SAS	SAS	SAS	SAS	SAS	COLD	COLD	SURVIVED	SURVIVED	SURVIVED
19	1227316	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
20	1230796	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
21	1235230	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
22	1235230	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
23	1171251	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
24	1222115	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
25	1242471	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
26	1242471	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
27	1230347	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
28	1232827	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
29	1240051	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
30	1246646	SAS	SAS	SAS	SAS	SAS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	SURVIVED	SURVIVED	SURVIVED
31	1018151	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
32	1249532	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
33	1250327	SAS	SAS	SAS	SAS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	SURVIVED	SURVIVED	SURVIVED
34	1227561	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED

S.NO	MRD.NO	OUTCOME OF FLAP AT 6 HRS	OUTCOME OF FLAP AT 12 HRS	OUTCOME OF FLAP AT 24 HRS	OUTCOME OF FLAP AT 48 HRS	LACTATE AT 0 HRS	LACTATE AT 1 HR	LACTATE AT 3 HRS	LACTATE AT 6 HRS	LACTATE AT 12 HRS	LACTATE AT 24 HRS	LACTATE AT 48 HRS	GLUCOSE AT 0 HR	GLUCOSE AT 1 HR
1	1130100	SURVIVED	SURVIVED	SURVIVED	SURVIVED	6.4	7.1	2	1.2	1.3	2	1.8	4.55	4.05
2	800878	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.9	4.8	8.2	7.1	4.3	7.1	4.8	12.2	7.74
3	1130100	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	15.4	10.2	18.2	-	-	-	-	1.5	3.33
4	646015	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4.6	1.8	2.4	2.8	4.4	2.6	2.2	10.1	8.88
5	1121162	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	3.1	3.7	3.6	4.2	4.6	7.5	3.2	6.16	6.83
6	1171751	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.2	6.8	4.2	6.8	5.2	1.4	1.8	7.43	8.38
7	1174116	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.8	2.4	4.6	8.3	9.7	5.2	3.2	8.55	7.9
8	1174808	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	5.6	7.6	8.2	12.6	-	-	-	8.65	7.4
9	1181144	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.4	2.6	3.2	3.2	2.1	2.3	1.8	7.89	7.1
10	1201101	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4.4	3.9	4.2	2.9	1.6	2	1.4	7.1	8.55
11	1158979	SURVIVED	SURVIVED	SURVIVED	SURVIVED	5.6	1.8	5.1	2.1	0.6	2.6	1.8	8.99	7.66
12	1208033	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.1	2.9	2.6	2.9	7.8	5.4	3.2	8.44	9.1
13	1173637	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4.6	3.2	2.3	2.1	2.1	1.6	1.8	8.21	8.99
14	1218930	SURVIVED	SURVIVED	SURVIVED	SURVIVED	7.1	7.5	6	6.5	6.2	3.4	2.8	11.16	9.66
15	1219875	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4.2	1.3	1.6	2.8	8.4	3.7	2.4	10.16	9.55
16	1221149	SURVIVED	SURVIVED	SURVIVED	SURVIVED	6.4	7.8	7.4	8.2	8.4	6.2	4.4	12.32	10.21
17	1216461	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4.2	3.7	2.8	1.6	1.3	1.6	2.4	9.55	7.78
18	1210101	SURVIVED	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	4.6	7.2	9.3	12.4	16.9	18.2	14.4	8.99	6.77
19	1227316	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4	5.4	7.6	11.5	7.3	4.2	3.6	8.5	1.11
20	1230796	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	8.2	9.8	13.6	10.1	8.8	9.8	8.6	4.22	4.88
21	1235230	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2	2.6	2.4	1.1	0.6	1.01	0.8	8.44	9.32
22	1235230	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.7	2.9	2.7	2.8	2.1	1.06	0.68	8.05	8.99
23	1171251	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.3	3.6	2.8	2.4	3.1	1.9	1.06	6.99	6.55
24	1222115	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	1.8	2	2.4	2	1.6	2.4	7.55	8.71
25	1242471	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4.7	3.3	4.2	4.6	3.9	2.6	2.9	7.44	6.16
26	1242471	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	2.1	3.2	2.3	1.7	1.6	0.6	7.88	6.16
27	1230347	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.4	3.7	4.2	3.2	1.1	0.6	0.7	6.94	8.21
28	1232827	SURVIVED	SURVIVED	SURVIVED	SURVIVED	1.6	2	2.1	1.4	1.2	0.9	1	8.55	7.88
29	1240051	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.9	3	3.3	4.1	4.5	4	3.1	8.05	7.88
30	1246646	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	4.8	7.9	5.2	8.8	16.5	-	-	6.66	6.55
31	1018151	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	1.01	3.6	3.8	4.2	2.6	1.4	7.1	5.61
32	1249532	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.3	2.8	3.2	4.8	3.1	2.3	0.9	5.27	8.49
33	1250327	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	2.8	3.2	4.8	7.2	-	-	-	8.88	5.44
34	1227561	SURVIVED	SURVIVED	SURVIVED	SURVIVED	11.8	7	5	2.6	2.8	3.2	2.6	14.82	8.94

S.NO	MRD.NO	GLUCOSE AT 3 HRS	GLUCOSE AT 6 HRS	GLUCOSE AT 12 HRS	GLUCOSE AT 24 HRS	GLUCOSE AT 48 HRS	L/G RATIO AT 0 HRS	L/G RATIO AT 1 HRS	L/G RATIO AT 3 HRS	L/G RATIO AT 6 HRS	L/G RATIO AT 12 HRS	L/G RATIO AT 24 HRS	L/G RATIO AT 48 HRS
1	1130100	3.94	4.94	6.55	7.21	7.44	1.41	0.35	0.51	0.24	0.2	0.28	0.24
2	800878	5.6	8.38	7.83	8.88	7.74	0.23	0.62	1.45	0.85	0.55	0.79	0.62
3	1130100	1.11	-	-	-	-	10.2	3.06	18.2	-	-	-	-
4	646015	8.44	7.1	9.66	7.33	6.94	0.45	0.2	0.28	0.39	0.46	0.35	0.32
5	1121162	6.22	3.33	1.39	4.33	3.89	0.5	0.54	0.58	1.26	3.3	1.73	0.82
6	1171751	9.27	8.55	7.94	6.11	7.43	0.43	0.81	0.45	0.8	0.7	0.23	0.24
7	1174116	7.4	7.3	7.3	9.2	7.4	0.33	0.02	0.62	1.14	1.13	0.57	0.43
8	1174808	5.9	1.17	-	-	-	0.65	1.03	1.39	10.77	-	-	-
9	1181144	7.55	6.11	5.5	7.8	7.22	0.3	0.37	0.42	0.52	0.38	0.3	0.25
10	1201101	6.94	9.32	6.55	7.88	8.66	0.03	0.46	0.61	0.31	0.24	0.25	0.16
11	1158979	8.66	7.88	8.77	7.1	6.49	0.62	0.23	0.59	0.27	0.07	0.37	0.28
12	1208033	6.94	10.1	6.7	7.66	8.44	0.37	0.32	0.37	0.016	1.11	0.7	0.38
13	1173637	7.1	7.83	7.33	7.88	8.88	0.56	0.36	0.32	0.27	0.29	0.2	0.2
14	1218930	8.05	8.21	8.55	7.1	7.78	0.64	0.78	0.74	0.79	0.73	0.48	0.36
15	1219875	8.88	7.78	7.38	4.44	6.78	0.41	0.14	0.18	0.36	1.14	0.83	0.35
16	1221149	9.77	8.55	7.66	6.22	8.05	0.52	0.76	0.76	0.96	1.1	0.99	0.55
17	1216461	6.78	7.38	4.44	7.78	10.16	0.44	0.48	0.41	0.22	0.05	0.21	0.24
18	1210101	7.1	4.88	2.99	3.44	1.11	0.51	1.06	1.31	2.54	4.3	5.29	14.4
19	1227316	4.44	4.38	3.89	6.77	8.99	0.47	5.4	1.71	0.15	1.88	0.62	0.4
20	1230796	1.61	3.33	5.22	6.22	4.9	1.94	2.01	8.45	3.03	1.69	1.58	1.75
21	1235230	6.88	8.21	6.55	5.99	6.77	0.24	0.28	0.35	0.13	0.09	0.17	0.12
22	1235230	8.44	6.6	5.99	6.11	6.99	0.34	0.32	0.32	0.42	0.35	0.83	0.097
23	1171251	7.27	7.88	5.99	6.11	6.88	0.33	0.55	0.39	0.3	0.52	0.31	0.15
24	1222115	7.94	6.72	6.16	5.32	5.44	0.34	0.21	0.25	0.36	0.32	0.3	0.44
25	1242471	5.33	6.83	6.61	7.44	6	0.64	0.53	0.8	0.67	0.59	0.35	0.48
26	1242471	5.33	6.88	7.33	8.55	6.05	0.33	0.34	0.6	0.34	0.23	0.19	0.1
27	1230347	9.27	6.44	6.16	5.11	4.66	0.35	0.45	0.45	0.5	0.17	0.12	0.15
28	1232827	9.27	7.38	6.72	6.16	7.33	0.19	0.26	0.23	0.19	0.18	0.15	0.14
29	1240051	6.66	8.66	6.55	6.72	5.99	0.36	0.38	0.5	0.47	0.69	0.6	0.52
30	1246646	8.71	7.55	1.39	-	-	0.72	1.21	0.6	1.66	11.8	-	-
31	1018151	7.55	8.99	6.66	8.55	5.33	0.37	0.18	0.48	0.42	0.63	0.3	0.26
32	1249532	8.99	8.38	8.99	5.44	6.11	0.44	0.02	0.36	0.57	0.34	0.42	0.45
33	1250327	2.72	4.61	-	-	-	0.32	0.59	1.76	1.56	-	-	-
34	1227561	6.66	7.77	7.38	6.44	6.83	0.8	0.78	0.75	0.33	0.38	0.5	0.38

S.NO	MRD.NO	OUTCOME OF FLAP	FINAL RESULTS	INTERVENTION DONE	DURATION AFTER SURGERY(hrs)	Group	LGR Recorded at other than designated intervals
1	1130100	SURVIVED	SURVIVED	-	-	A	-
2	800878	SURVIVED	SURVIVED	-	-	A	-
3	1130100	ARTERIAL INSUFFICIENCY- FLAP DID NOT SURVIVE	NOT SURVIVED	REDO ANASTOMOSES	4	B	-
4	646015	SURVIVED	SURVIVED	-	-	A	-
5	1121162	VENOUS INSUFFICIENCY-MARGINAL NECROSIS	PARTIAL NECROSIS	RUSHED D25% AND REMOVAL OF SUTURE	30	B	-
6	1171751	SURVIVED	SURVIVED	-	-	A	AT 18 HRS.L/G= 0.38
7	1174116	SURVIVED	SURVIVED	-	-	A	-
8	1174808	VENOUS INSUFFICIENCY- FLAP DID NOT SURVIVE	NOT SURVIVED	FLAP REMOVED SURGICALLY	8	B	-
9	1181144	SURVIVED	SURVIVED	-	-	A	-
10	1201101	SURVIVED	SURVIVED	-	-	A	-
11	1158979	SURVIVED	SURVIVED	-	-	A	-
12	1208033	SURVIVED	SURVIVED	-	-	A	-
13	1173637	SURVIVED	SURVIVED	-	-	A	-
14	1218930	SURVIVED	SURVIVED	-	-	A	-
15	1219875	SURVIVED	SURVIVED	-	-	A	AT 16HRS L/G=0.52
16	1221149	SURVIVED	SURVIVED	-	-	A	-
17	1216461	SURVIVED	SURVIVED	-	-	A	-
18	1210101	VENOUS INSUFFICIENCY-MARGINAL NECROSIS	PARTIAL NECROSIS	SCRATCHING & HEPARIN GAUZE PLACEMENT	7	B	AT 16 HRS L/G=20.4; AT 28 HRS- 4.78
19	1227316	SURVIVED	SURVIVED	-	-	A	BODY RBS 2.89 AT 1 HR. D25 rushed. At 2 hrs L/G= 1.52; AT 8 HRS L/G= 1.72
20	1230796	VENOUS INSUFFICIENCY-MARGINAL NECROSIS	PARTIAL NECROSIS	SCRATCHING & HEPARIN GAUZE PLACEMENT	2	B	L/G 4H,5HR,8HR = 17.7, 12.43, 1.73 RESPECTIVELY
21	1235230	SURVIVED	SURVIVED	-	-	A	-
22	1235230	SURVIVED	SURVIVED	-	-	A	-
23	1171251	SURVIVED	SURVIVED	-	-	A	-
24	1222115	SURVIVED	SURVIVED	-	-	A	-
25	1242471	SURVIVED	SURVIVED	-	-	A	-
26	1242471	SURVIVED	SURVIVED	-	-	A	-
27	1230347	SURVIVED	SURVIVED	-	-	A	-
28	1232827	SURVIVED	SURVIVED	-	-	A	-
29	1240051	SURVIVED	SURVIVED	-	-	A	-
30	1246646	ARTERIAL INSUFFICIENCY- MARGINAL NECROSIS	PARTIAL NECROSIS	REDO ANASTOMOSES	12	B	L/G AT 8, 10 HRS = 2.33, 4.23 RESPECTIVELY
31	1018151	SURVIVED	SURVIVED	-	-	A	-
32	1249532	SURVIVED	SURVIVED	-	-	A	-
33	1250327	VENOUS INSUFFICIENCY- FLAP SURVIVED	SURVIVED	Suture removal, distally & laterally	6	B	L/G AT 8 HRS = 1.04
34	1227561	SURVIVED	SURVIVED	-	-	A	-

S.NO	MRD.NO	NAME	Age	SEX (M/F)	CAUSE OF DEFECT	WOUND SITE	FLAP NAME	FLAP TYPE	COLOUR 0 HRS	COLOUR 1 HRS	COLOUR 3 HRS	COLOUR 6 HRS	COLOUR 12 HRS
35	1222115	R	25	M	PRESSURE SORE	TROCHANTER	TFL MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
36	421470	NK	48	M	CARCINOMA	SACRUM	TFL MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
37	1247930	GS	24	M	POST ELECTRIC BURN	HAND	ABDOMINAL FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
38	927694	MK	34	F	CARCINOMA	BREAST	LATISSIMUS DORSI MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
39	1249344	MS	50	M	POST TRAUMATIC	LEG	CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	BLUISH
40	1249420	L	43	F	EXTRAVASATION INJURY	HAND	HYPOGASTRIC FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
41	1256915	JS	31	M	POST TRAUMATIC	LEG	CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
42	1267059	KR	55	M	HEAD & NECK CANCER	ORAL CAVITY	PECTORALIS MAJOR MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
43	1267666	SS	72	M	HEAD & NECK CANCER	ORAL CAVITY	PECTORALIS MAJOR MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
44	1263896	JS	38	M	POST ELECTRIC BURN	FOREARM	GROIN FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
45	1278004	R	24	M	POST TRAUMATIC	HAND	GROIN FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
46	1272734	JS	31	M	POST TRAUMATIC	LEG	FASCIOCUTANEOUS TRANSPOSITION FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
47	1284905	NS	55	M	POST TRAUMATIC	ANKLE/FOOT	REVERSE SURAL ARTERY FLAP	PEDICLED	SAS	BLUISH	BLUISH	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
48	1305947	JS	26	M	POST TRAUMATIC	LEG	FASCIOCUTANEOUS TRANSPOSITION FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
49	1310659	UR	52	F	HEAD & NECK CANCER	ORAL CAVITY	PECTORALIS MAJOR MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
50	1307999	KK	47	M	POST TRAUMATIC	HAND	GROIN FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
51	1298962	GS	18	M	POST TRAUMATIC	LEG	CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
52	1304213	DS	40	M	POST TRAUMATIC	LEG	CROSS LEG FASCIOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
53	1311645	TS	37	M	POST ELECTRIC BURN	FACE	ALT FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
54	1018151	GS	43	M	HEAD & NECK CANCER	ORAL CAVITY	LATISSIMUS DORSI MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
55	1358051	RA	48	M	HEAD & NECK CANCER	ORAL CAVITY	LATISSIMUS DORSI MYOCUTANEOUS FLAP	PEDICLED	SAS	SAS	SAS	SAS	SAS
56	1385924	F	38	F	HEAD & NECK CANCER	FACE	ALT FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
57	1379067	GS	46	M	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
58	1403148	JK	39	F	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
59	1376295	RR	51	M	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
60	1414375	NS	54	M	POST TRAUMATIC	ANKLE/FOOT	ALT FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
61	1418618	HB	55	M	HEAD & NECK CANCER	ORAL CAVITY	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
62	1434269	BD	52	M	HEAD & NECK CANCER	FACE	RADIAL ARTERY FOREARM FREE FLAP	FREE	SAS	SAS	SAS	SAS	SAS
63	1406135	I	49	M	HEAD & NECK CANCER	ORAL CAVITY	FREE FIBULA OSTEOCUTANEOUS FLAP	FREE	SAS	SAS	SAS	SAS	SAS

S.NO	MRD.NO	COLOUR 24 HRS	COLOUR 48HRS	CAPILLARY REFILL 0 HRS	CAPILLARY REFILL 1 HRS	CAPILLARY REFILL 3 HRS	CAPILLARY REFILL 6 HRS	CAPILLARY REFILL 12 HRS	CAPILLARY REFILL 24 HRS
35	1222115	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
36	421470	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
37	1247930	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
38	927694	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
39	1249344	BLUISH	BLUISH	WNL	WNL	WNL	WNL	SPONTANEOUS	SPONTANEOUS
40	1249420	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
41	1256915	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
42	1267059	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
43	1267666	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
44	1263896	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
45	1278004	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
46	1272734	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
47	1284905	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	WNL	SPONTANEOUS	ABSENT	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
48	1305947	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
49	1310659	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
50	1307999	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
51	1298962	BLUISH	BLUISH	WNL	WNL	WNL	WNL	WNL	ABSENT
52	1304213	BLUISH	BLUISH	WNL	WNL	WNL	WNL	WNL	ABSENT
53	1311645	BLUISH	BLUISH	WNL	WNL	WNL	WNL	WNL	ABSENT
54	1018151	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
55	1358051	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
56	1385924	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
57	1379067	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
58	1403148	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
59	1376295	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
60	1414375	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
61	1418618	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
62	1434269	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL
63	1406135	SAS	SAS	WNL	WNL	WNL	WNL	WNL	WNL

S.NO	MRD.NO	CAPILLARY REFILL 48 HRS	BLEEDING CHARACTERISTICS AT 0 HRS	BLEEDING CHARACTERISTICS AT 1 HRS	BLEEDING CHARACTERISTICS AT 3 HRS	BLEEDING CHARACTERISTICS AT 6 HRS	BLEEDING CHARACTERISTICS AT 12 HRS	BLEEDING CHARACTERISTICS AT 24 HRS	BLEEDING CHARACTERISTICS AT 48 HRS
35	1222115	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
36	421470	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
37	1247930	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
38	927694	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
39	1249344	SPONTANEOUS	ARTERIAL	ARTERIAL	ARTERIAL	VENOUS	VENOUS	VENOUS	VENOUS
40	1249420	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
41	1256915	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
42	1267059	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
43	1267666	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
44	1263896	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
45	1278004	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
46	1272734	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
47	1284905	NOT SURVIVED/MARGINAL NECROSIS	ARTERIAL	VENOUS	NO BLEED	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS
48	1305947	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
49	1310659	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
50	1307999	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
51	1298962	ABSENT	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	NO BLEED	NO BLEED
52	1304213	ABSENT	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	NO BLEED
53	1311645	ABSENT	ARTERIAL	ARTERIAL	VENOUS	VENOUS	VENOUS	NO BLEED	NO BLEED
54	1018151	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
55	1358051	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
56	1385924	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
57	1379067	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
58	1403148	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
59	1376295	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
60	1414375	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
61	1418618	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
62	1434269	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL
63	1406135	WNL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL	ARTERIAL

S.NO	MRD.NO	TEMPERATURE AT 0 HRS	TEMPERATURE AT 1 HRS	TEMPERATURE AT 3 HRS	TEMPERATURE AT 6 HRS	TEMPERATURE AT 12 HRS	TEMPERATURE AT 24 HRS	TEMPERATURE AT 48 HRS	OUTCOME OF FLAP AT 0 HRS	OUTCOME OF FLAP AT 1 HRS	OUTCOME OF FLAP AT 3 HRS
35	1222115	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
36	421470	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
37	1247930	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
38	927694	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
39	1249344	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
40	1249420	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
41	1256915	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
42	1267059	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
43	1267666	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
44	1263896	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
45	1278004	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
46	1272734	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
47	1284905	SAS	SAS	SAS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	NOT SURVIVED/MARGINAL NECROSIS	SURVIVED	SURVIVED	SURVIVED
48	1305947	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
49	1310659	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
50	1307999	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
51	1298962	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
52	1304213	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
53	1311645	SAS	SAS	SAS	SAS	SAS	COLD	COLD	SURVIVED	SURVIVED	SURVIVED
54	1018151	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
55	1358051	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
56	1385924	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
57	1379067	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
58	1403148	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
59	1376295	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
60	1414375	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
61	1418618	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
62	1434269	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED
63	1406135	SAS	SAS	SAS	SAS	SAS	SAS	SAS	SURVIVED	SURVIVED	SURVIVED

S.NO	MRD.NO	OUTCOME OF FLAP AT 6 HRS	OUTCOME OF FLAP AT 12 HRS	OUTCOME OF FLAP AT 24 HRS	OUTCOME OF FLAP AT 48 HRS	LACTATE AT 0 HRS	LACTATE AT 1 HR	LACTATE AT 3 HRS	LACTATE AT 6 HRS	LACTATE AT 12 HRS	LACTATE AT 24 HRS	LACTATE AT 48 HRS	GLUCOSE AT 0 HR	GLUCOSE AT 1 HR
35	1222115	SURVIVED	SURVIVED	SURVIVED	SURVIVED	8.6	9.2	8.8	10	9	6.7	5.4	8.99	8.49
36	421470	SURVIVED	SURVIVED	SURVIVED	SURVIVED	6.8	6.3	9.5	2.7	10.8	6	9.1	9.27	13.93
37	1247930	SURVIVED	SURVIVED	SURVIVED	SURVIVED	5.6	7.9	6.8	8.3	6.2	5.5	2.6	12.21	10.71
38	927694	SURVIVED	SURVIVED	SURVIVED	SURVIVED	1.6	6.3	11.5	5.4	3.5	4.5	3.1	10.43	9.21
39	1249344	SURVIVED	SURVIVED	SURVIVED	MARGINAL NECROSIS	9.3	8.2	9.7	11.9	15.7	13.1	12.6	8.71	7.88
40	1249420	SURVIVED	SURVIVED	SURVIVED	SURVIVED	5.6	4.9	6.2	5.1	3	3.2	2.9	11.99	8.94
41	1256915	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.2	5.4	2.9	2.6	2.9	1.8	3.1	6.55	8.66
42	1267059	SURVIVED	SURVIVED	SURVIVED	SURVIVED	4.8	4	2.9	5.2	3.2	2.8	1.9	7.74	8.88
43	1267666	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	3.1	4.2	2.9	2.6	3.1	1.9	7.78	6.78
44	1263896	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	3.2	4.5	3.9	4.8	5.6	7.8	8.66	5.44
45	1278004	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	1.8	2.4	1.6	3.2	2.9	1.8	8.66	7.38
46	1272734	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.6	2.4	1.6	1.9	2	2.8	1.8	7.88	6.88
47	1284905	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	MARGINAL NECROSIS	11.7	17.6	18.2	-	-	-	-	6.16	1.11
48	1305947	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.2	2.4	1.8	1.6	2	1.4	1.2	8.22	8.58
49	1310659	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	1.8	2	3.4	0.6	1	1.2	6.45	6.77
50	1307999	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.6	4.2	5	2.8	1.6	0.6	1.2	6.22	5.99
51	1298962	SURVIVED	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	3.2	4.6	5.4	6.8	14.4	-	-	7	7.33
52	1304213	SURVIVED	SURVIVED	SURVIVED	MARGINAL NECROSIS	4.8	3.6	3	2.9	14.2	12.6	-	6.11	5.22
53	1311645	SURVIVED	SURVIVED	MARGINAL NECROSIS	MARGINAL NECROSIS	8.9	7.8	14.2	19.9	18.2	-	-	6.11	5.33
54	1018151	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.2	4.8	2.6	1.6	0.8	0.4	2.2	8.55	7.33
55	1358051	SURVIVED	SURVIVED	SURVIVED	SURVIVED	9.1	1.1	4.4	5.1	4	3.7	2.9	15.21	15.6
56	1385924	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.2	2.1	3.4	2.6	1.9	0.6	2.8	6.44	6.88
57	1379067	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.6	3.3	4.2	3.2	2.8	2	3.4	10.1	10.77
58	1403148	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.2	4.2	3.6	4.9	5.2	4.6	3.9	6.99	7.33
59	1376295	SURVIVED	SURVIVED	SURVIVED	SURVIVED	5.2	4.8	3.3	2.7	4.5	3.1	2.8	7.38	8.44
60	1414375	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.2	1.8	2.6	3	3.3	1.9	2.1	8.05	10.93
61	1418618	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.2	4.1	3.6	2.4	2.8	1.6	0.9	6.22	7.33
62	1434269	SURVIVED	SURVIVED	SURVIVED	SURVIVED	3.2	2.6	2.1	2.2	2	3.2	1.8	7.33	8.05
63	1406135	SURVIVED	SURVIVED	SURVIVED	SURVIVED	2.6	3.9	4.1	3.6	2.2	2.4	1.8	8.05	7.33

S.NO	MRD.NO	GLUCOSE AT 3 HRS	GLUCOSE AT 6 HRS	GLUCOSE AT 12 HRS	GLUCOSE AT 24 HRS	GLUCOSE AT 48 HRS	L/G RATIO AT 0 HRS	L/G RATIO AT 1 HRS	L/G RATIO AT 3 HRS	L/G RATIO AT 6 HRS	L/G RATIO AT 12 HRS	L/G RATIO AT 24 HRS	L/G RATIO AT 48 HRS
35	1222115	5.44	5.77	7.22	8.05	7.66	0.96	1.08	1.62	1.73	1.25	0.83	0.7
36	421470	11.16	11.16	9.32	9.55	3.83	0.73	0.45	0.85	0.24	1.16	0.63	2.37
37	1247930	8.05	11.99	12.21	10.1	8.55	0.46	0.74	0.84	0.69	0.51	0.54	0.3
38	927694	7.38	9.21	9.82	8.32	7.16	0.15	0.68	1.56	0.59	0.36	0.54	0.43
39	1249344	7.33	6.55	7.38	19.31	15.6	1.07	1.04	1.32	1.82	2.13	0.68	0.81
40	1249420	6.11	5.44	10.1	6.66	8.55	0.47	0.55	1.01	0.94	0.3	0.48	0.34
41	1256915	8.55	7.88	6.94	6.7	7.66	0.49	0.62	0.34	0.33	0.42	0.27	0.4
42	1267059	12.2	9.2	7.4	7.3	6.55	0.62	0.45	0.23	0.57	0.43	0.38	0.29
43	1267666	7.74	8.88	7.1	7.33	7.4	0.33	0.46	0.54	0.33	0.37	0.42	0.26
44	1263896	6.99	7.71	8.99	0.05	5.77	0.3	0.59	0.64	0.51	0.53	0.79	1.35
45	1278004	7.16	7.88	5.33	7.99	6.44	0.3	0.24	0.34	0.2	0.6	0.36	0.28
46	1272734	5.99	6.11	7.55	5.44	8.71	0.46	0.35	0.27	0.31	0.26	0.52	0.21
47	1284905	-	-	-	-	-	1.9	17.6	-	-	-	-	-
48	1305947	6.55	6.71	5.94	5.72	5.28	0.27	0.28	0.27	0.24	0.34	0.24	0.23
49	1310659	6.05	7.33	8.55	7.33	5.66	0.4	0.26	0.33	0.46	0.07	0.14	0.21
50	1307999	5.22	6.99	6.6	5.66	5.44	0.58	0.7	0.96	0.4	0.24	0.11	0.22
51	1298962	6.44	5.22	2	-	-	0.46	0.63	0.84	1.3	7.2	-	-
52	1304213	6.44	6.16	4.72	5	-	0.79	0.69	0.47	0.47	3.01	2.52	-
53	1311645	4.55	3.55	2.88	-	-	1.46	1.28	2.66	5.61	6.32	-	-
54	1018151	6.83	7.27	6.11	6.22	6.44	0.37	0.65	0.38	0.22	0.13	0.06	0.34
55	1358051	14.93	19.37	20.81	7.94	7.44	0.6	0.07	0.29	0.26	0.19	0.47	0.39
56	1385924	8.33	7.38	8.21	5.99	6.33	0.5	0.31	0.41	0.35	0.23	0.1	0.44
57	1379067	10.43	8.94	8.21	7.88	7.55	0.36	0.31	0.4	0.36	0.34	0.25	0.45
58	1403148	7.77	7.38	7.1	7.55	6.22	0.46	0.57	0.46	0.66	0.73	0.61	0.63
59	1376295	6.11	6.55	5.33	5.77	6.49	0.7	0.57	0.54	0.41	0.84	0.54	0.43
60	1414375	7.1	7.55	6.11	6.16	7.33	0.27	0.16	0.37	0.4	0.54	0.31	0.29
61	1418618	7.77	7.49	6.99	6.05	6.83	0.51	0.56	0.46	0.32	0.4	0.26	0.13
62	1434269	7.55	6.77	7.88	7.33	6.83	0.44	0.32	0.28	0.32	0.25	0.44	0.26
63	1406135	6.22	6.77	6.05	7.22	7.16	0.32	0.53	0.66	0.53	0.36	0.33	0.25

S.NO	MRD.NO	OUTCOME OF FLAP	FINAL RESULTS	INTERVENTION DONE	DURATION AFTER SURGERY(hrs)	Group	LGR Recorded at other than designated intervals
35	1222115	SURVIVED	SURVIVED	-	-	A	L/G at 10 hrs= 1.13
36	421470	SURVIVED	SURVIVED	-	-	A	-
37	1247930	SURVIVED	SURVIVED	-	-	A	-
38	927694	SURVIVED	SURVIVED	-	-	A	-
39	1249344	VENOUS INSUFFICIENCY-MARGINAL NECROSIS	PARTIAL NECROSIS	-	-	B	L/G AT 14, 18 HRS = 2.78, 1.51 RESPECTIVELY
40	1249420	SURVIVED	SURVIVED	-	-	A	-
41	1256915	SURVIVED	SURVIVED	-	-	A	-
42	1267059	SURVIVED	SURVIVED	-	-	A	-
43	1267666	SURVIVED	SURVIVED	-	-	A	-
44	1263896	SURVIVED	SURVIVED	-	-	A	-
45	1278004	SURVIVED	SURVIVED	-	-	A	-
46	1272734	SURVIVED	SURVIVED	-	-	A	-
47	1284905	VENOUS INSUFFICIENCY- FLAP SURVIVED	SURVIVED	Sutures removed, NPWT Applied	3	B	L/G AT 2 HRS- NO DROP
48	1305947	SURVIVED	SURVIVED	-	-	A	-
49	1310659	SURVIVED	SURVIVED	-	-	A	-
50	1307999	SURVIVED	SURVIVED	-	-	A	-
51	1298962	ARTERIAL INSUFFICIENCY- MARGINAL NECROSIS	PARTIAL NECROSIS	-	-	B	-
52	1304213	ARTERIAL INSUFFICIENCY- MARGINAL NECROSIS	PARTIAL NECROSIS	FLAP READJUSTMENT AFTER 48 HRS	-	B	L/G AT 14, 16, 20HR = 2.9,3.26,2.67 RESPECTIVELY
53	1311645	VENOUS INSUFFICIENCY- FLAP DID NOT SURVIVE	NOT SURVIVED	FLAP REMOVED SURGICALLY	26	B	L/G AT 5 HRS = 4.41
54	1018151	SURVIVED	SURVIVED	-	-	A	-
55	1358051	SURVIVED	SURVIVED	-	-	A	-
56	1385924	SURVIVED	SURVIVED	-	-	A	-
57	1379067	SURVIVED	SURVIVED	-	-	A	-
58	1403148	SURVIVED	SURVIVED	-	-	A	-
59	1376295	SURVIVED	SURVIVED	-	-	A	-
60	1414375	SURVIVED	SURVIVED	-	-	A	-
61	1418618	SURVIVED	SURVIVED	-	-	A	-
62	1434269	SURVIVED	SURVIVED	-	-	A	-
63	1406135	SURVIVED	SURVIVED	-	-	A	-

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Date 05/11/21

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Renu Verma**, a student of your college for **MCh Plastic & Reconstructive Surgery** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

Role of capillary lactate/glucose ratio from flap to predict flap survival.

Supervisor
Dr. Rajinder Mittal
Prof. & Head,
Dept. of Plastic Surgery,
DMCH, Ludhiana.

Co-Supervisor

Dr. Sheerin Shah
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Dept. of Plastic Surgery,
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The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

179. If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.

180. The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.

ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

M.Ch

IN

PLASTIC AND RECONSTRUCTIVE SURGERY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

AUGUST, 2021

DR. RENU VERMA

DEPARTMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY
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LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL
For the degree of	M.Ch Plastic and Reconstructive Surgery
Name of the candidate	Dr. Renu Verma
Supervisor	Dr. Rajinder Mittal, Professor & Head, Department of Plastic and Reconstructive Surgery, DMCH, Ludhiana
Co-supervisor	Dr. Sheerin Shah, Associate Professor, Department of Plastic and Reconstructive Surgery , DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
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Introduction: Flaps are the workhorse of Reconstructive and microvascular surgery. The visible improvement in the success rate of flaps is attributed to several reliable flaps monitoring tools. Clinical assessment is still the gold standard which utilizes subjective parameters such as temperature, colour, turgor and capillary refill and hence may be faulty. Several objective methods have thus evolved over time. Amongst them, flap glucose and lactate measurements as a flap monitoring tool for predicting flap survival and need for re-exploration is more objective, simpler, need no expertise and are cost effective.

Aim of Study: To study the role of capillary Lactate/Glucose ratio as a predictor of Flap Survival.

Materials & Methods: A Prospective study will be conducted on all consenting patients fulfilling the inclusion criteria, who will undergo any kind of flap surgery in the Department of Plastic Surgery, Dayanand Medical College and Hospital, Ludhiana from August 2021 to December 2022. After the surgery, flap Lactate/Glucose ratio will be calculated and recorded at 0, 1,3,6,12,24 and 48 hrs. Measurements will also be done whenever flap will show any signs of compromised vascularity in between these intervals.

Two groups will be made:

Group 1: Flaps with no post-operative complications

Group 2: Flap with post-operative complications (signs of necrosis, need of any surgical intervention)

The L/G ratio of 2 groups will be compared and data will be utilized to calculate the cut off value of L/G ratio at which flaps begin to show signs of non-viability.

Clinical Significance: To study the role of capillary Lactate/Glucose ratio as a predictor of Flap Survival.

Keywords: Flap Monitoring, Objective tool, Lactate/Glucose ratio, cost effective.

(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-Supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL"** exist in the Department of Plastic and Reconstructive surgery, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

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Place : Ludhiana

Date : 28/08/21

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
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Name of candidate	Dr. Renu Verma
Department	Plastic and Reconstructive Surgery
Topic of Thesis	ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL
Likely date of appearing in PG examination	May/June- 2023
Date of enrollment	28.12.2020
Name of Head of Department	Dr. Rajinder Mittal
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Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
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	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

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Approved: Yes / ~~No~~

Prof. Arvind Malhotra
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Ethics Committee

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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

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3	COURSE OF STUDY AND SUBJECT	M.Ch. in Plastic and Reconstructive Surgery
4	DATE OF ADMISSION OF COURSE	28/12/20
5	TITLE OF THE TOPIC	ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL

6.0 BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR STUDY

Flaps are the workhorse of Reconstructive and microvascular surgery. A flap is a unit of tissue which is transferred from a donor to a recipient site with its blood supply.[1] Numerous classification schemes exist. Flaps can be characterized by their component parts (cutaneous, musculocutaneous, osseocutaneous), their special relationship with the defect (local, regional distant or free), the nature of blood supply (random or axial), and finally by the movement that is required to fill the desired defect (advancement, pivot, transposition or interpolation). [1]

Optimal blood supply is essential for flaps to survive, and compromised supply, if detected early, can help salvage a dying flap.[2]

Minimizing flap failure is a key goal for any Reconstructive and Microsurgical unit.

Veins, due to their thin walls and fragile structure are more susceptible to injury as compared to arteries. It is well known that congestion due to venous thrombosis is more common and congested flaps are more prone to fail than arterially compromised flaps.[3]

Data suggest that the commonest cause of free flap failure is a problem with the venous anastomosis, occurring within the first 24 to 48 hours postoperatively.[4,5]

Their may be conditions like vasospasm that are self limiting or temporary and may be resolved by local warming or pain relief but certain disturbances in the flow of blood like external pressure or low perfusion pressure if prolonged can lead to venous occlusion which if prolonged can impair outflow, ultimately leading to thrombus formation. During this ongoing process, flap may clinically look normal to an unexperienced eye.

The success of a free flap salvage operation is dependent on the early detection of vascular compromise and the subsequent short take-back time to the operating room.[6]

In case of pedicled flaps, the causes may be hematoma, external compression by tissue odema or tight Dressing, twisting of pedicle, and

thrombosis leading to venous congestion which if prolonged can lead to flap necrosis.

The visible improvement in the success rate of flaps is attributed to the advancement of microsurgical techniques, instruments and invention of several reliable flaps monitoring tools. An ideal monitoring method should be harmless to the patient and the flap, accurate and reliable, capable of providing rapid response to circulatory change, capable of being used by all personnel, reproducible, inexpensive, and portable.[7]

There are several methods of flap monitoring that have been described. Clinical assessment is still the gold standard which utilizes parameters such as temperature, colour, turgor and capillary refill. These are all the subjective parameters and may be faulty in case of inexperience or factors like inadequate lighting or poor visibility. Several objective methods have thus evolved over time- Doppler ultrasonography of the vascular pedicle[8], measurement of tissue oxygenation[9], analysis by microdialysis[10], use of intra-arterial or intravenous catheters[11], and quantitative spectral imaging (which measures distance, oxyhemoglobin, deoxyhemoglobin, total hemoglobin, and tissue saturation)[12], tissue pH monitoring, skin temperature probes, near infrared spectroscopy, photoplethysmography as well as the use of fluorescent dyes amongst many others. These methods require complex apparatus and are expensive.

Mechanism of Microdialysis is based on monitoring the metabolic changes in the flap by detecting changes in glucose, lactate and pyruvate much before they are actually reflected in the physiological parameters or in peripheral blood.

Tachi et al said tissue glucose level decreases earlier and faster than body glucose level. They used continuous tissue glucose measuring device and hypothesized that when venous congestion occurs, the tissue becomes hypoxic, and anaerobic glycolysis is accelerated, resulting in tissue glucose depletion.[13]

Based on these, several studies have utilized flap glucose and lactate measurements as a flap monitoring tool for predicting flap survival and

need for re-exploration. These are more objective, simpler, need no expertise and are cost effective.

The aim of our study is to compare the clinical assessment and intracapillary Lactate to Glucose ratio in predicting flap survival.

6.2 REVIEW OF LITERATURE

Flap monitoring is very significant to a plastic and reconstructive surgeon. The survival of the flap (free as well as pedicled) and the success of the surgeon depends not only upon the surgical technique and the experienced hands but also the post-op flap monitoring and timely decision to re-explore in addition the general condition of the patient.

Though the success rate of free flap is reported to be 95% or higher, the problem of anastomosed artery or vein remains a major cause of tissue loss[7] and in general, venous compromise or congestion is more common cause of flap failure than arterial thrombosis other causes being pedicle complications such as extrinsic compression or kinking.

Most of these complications usually occurs within 48 hours of surgery, thus demands early detection followed by timely intervention before permanent damage sets in. This needs close monitoring of flaps.

Although Clinical Evaluation still remains the gold standard which include Colour, temperature, turgor, Capillary refill time and pin prick to check the colour and character of bleed but this is a subjective observation and requires experience. But it is often too late when these clinical signs become apparent.[14] Various other objective methods are available which are not cost effective.[8-12] Amongst them, the microdialysis which analyses metabolic changes in flap, has shown that Capillary glucose level drops at the time of congestion or ischemia. Within flap blood glucose measurement cannot be relied upon for ischemic flaps, as sufficient blood cannot be obtained to measure the glucose level in a pinprick test. Dynamics of blood glucose concentration in flaps during the postoperative period is such that a single reading of flap's blood glucose is not a reliable indicator of vascular compromise. However, mean of repeated measurements of flap's blood glucose levels taken at different time intervals can help in deducing the venous compromise.[14] Exact mechanisms responsible are unclear. Sakakibara et al[15], in 2010 hypothesized that in venous thrombosis, flap's blood glucose concentration is reduced due to continual uptake of glucose from the stagnated blood. Venous

thrombosis might decrease the inflow of blood from flap's artery by increasing venous and capillary pressures. Lack of adequate blood flow may cause continued uptake of glucose from a slow-moving blood resulting in a local hypoglycemic state. Further, reduced blood flow leads to consequent increase in anaerobic metabolism. The anaerobic pathway uses glucose inefficiently, which further worsens local hypoglycemia.[16]

In 2010, Sakakibara et al. [15] reported a lower blood glucose level in congestive flaps in clinical cases.

In 2011, Hara et al [14] described blood glucose measurement for flap monitoring and reported that the blood glucose level of 62mg/dl is the best cutoff value for detecting venous thrombosis. They noted the Blood Glucose in 33 free or pedicled tissue transfers over time postoperatively. Partial necrosis of the vascular territory was found at 5 points (9%), and blood flow disorder due to a venous thrombus was found at 5 points (9%). The mean blood glucose level in the congestive flaps was significantly lower than that in healthy flaps. ROC curve analysis was used to determine a cutoff value for Blood Glucose Monitoring of 62 mg/dL, at which the sensitivity and specificity were 88% and 82%, respectively ($p < 0.0001$). It was concluded that Blood Glucose Monitoring was an easy and accessible adjunct to flap monitoring, likely to reduce postoperative complications caused by the development of a venous thrombus after free tissue transplantation.

In 2015, Bashir et al[17] determine the diagnostic accuracy of blood glucose measurements within flaps in early detection of venous compromise. 127 pedicled and free flaps, including replants, performed on patients between 12 and 60 years of age. Within flap blood glucose measurements were performed using pinprick and a blood glucometer at 0, 6, 12, 24 and 48 hours after operation. Daily examination for clinical signs of venous compromise was used to determine flap viability for up to 7th day after operation. A cut-off value of 62 mg/dL was determined using a receiver operating characteristic curve. Using this cut-off value, the sensitivity and positive predictive values of within flap blood glucose for determining venous compromise were 90% and

91%, respectively, whereas the specificity and negative predictive values were 78% and 76%, respectively. The overall diagnostic accuracy of within flap blood glucose was 87%.

In 2016, A study done by Muthulakshmi et al[18] on 50 patients who underwent BK amputations, 5 patients developed postoperative flap necrosis due to venous thrombosis. Flap blood glucose level was measured at 0,6,24 hours. Blood glucose levels <62 mg/dl , for detection of flap survival had a sensitivity, specificity and positive predictive value of 86.66%, 100%, at 0 hour, 100%, 100%, 100% at 6 hours and 97.77%, 100%, 100% at 24 hours respectively. The study confirmed a decrease in capillary glucose in all 5 flaps which underwent necrosis and gangrene in the first 24 hours after surgery.

Several reports described the effects of blood flow obstruction on blood glucose and lactate values. In 1989, Heden and Sollevi [19] showed that tissue hypoxia caused lactate values to increase and blood glucose values to decrease at the same time during ischemia.

In 1998, Rojdmark et al [20] measured intra-flap blood glucose and lactate values to evaluate flap blood flow. Five patients provided with free flaps, were included in the study. Microdialysis catheters were placed in the flap and in normal control tissue. Laser Doppler flowmetry was used postoperatively to verify circulatory changes. During flap transfer, and the ischemia that was induced by the operation, the glucose concentration in the interstitial flap tissue decreased substantially, whereas the lactate and glycerol concentrations both increased. When vascular anastomoses had been completed these three parameters tended to return to preoperative levels. This metabolic pattern was not seen in the control tissue, thus suggesting that it may be typical for local ischemia. In two of the patients the pattern reappeared in connection with postoperative venous thrombosis, and disappeared when the thromboses were surgically removed.

In 2008, Sorensen [21] used those values for free jejunum flaps. His was a retrospective study of 14 patients undergoing resection of carcinoma in the upper part of the esophagus and reconstruction with a free

jejunal flap. The metabolism in all 14 jejunal transfers was monitored by use of microdialysis. The data were analyzed looking for reliable parameters detecting critical ischemia. Critical ischemia was suspected in two cases. Both of these cases were surgically revised, ischemia in the jejunal flap was verified, and the jejunal flaps were revascularized. All 14 jejunal flaps survived. It was reported that using the concentration of glucose in the microdialysate, it was possible to detect the two cases of critical ischemia. Yet, the most reliable parameter seemed to be the retrospectively calculated lactate:glucose ratio; in both the ischemic flaps, the lactate:glucose ratio exceeded more than 1000% the maximum values found in all the nonischemic flaps.

In 2006, Setala et al [22] reported that blood glucose values decreased 2 to 8 hours before clinical findings emerged, and lactate values increased 1 to 12 hours before the findings of congestion. They speculated that the tissue was in a transient anaerobic metabolic state until the post-reperfusion flap tissue adapted to the new perfusion pattern. Unlike lactate values, after the blood glucose values increased to higher than the upper limit for temporary decreases, they remained higher than the reference value.

In 2013, a study used microdialysis in monitoring 268 microsurgical flaps and compared the metabolic data of normally perfused flaps to those that suffered from relative or absolute perfusion failure. It was found that tissue glucose and lactate concentrations and especially their relation (lactate-to-glucose ratio) can show the presence of ischemia and aid in decision making, whether to reoperate or not. High lactate-to-glucose ratio may also predict total or partial flap necrosis. If L/G ratio was 1.0, the flap was safe. If the level reached 5.0, still only 8% suffered from some kind of difficulty but when the level of 22 was reached, the ischemia was usually evident.[23]

In 2014, Henault et al.[24] reported a study in which measurements of capillary glucose and lactate in the flaps were done. These parameters were compared to standardized clinical monitoring during the first five days. Two sets of data (eventful versus uneventful postoperative period) were analyzed to define the thresholds of lactate and glucose

values for diagnosis of pedicle complications, and to establish parameters for this screening test. Over a period of 19 months, 37 patients were included. With 5 pedicle impairments, complication thresholds were defined as lactate >= 6.4 mmol/L and glucose <= 3.85 mmol/L , in order to obtain a sensitivity of 98.5% and a specificity of 99.5% for the test. Modifications of capillary glucose and lactate measurements appeared in average 5.7 hours earlier than clinical symptoms in pedicle impairments. The mean cost of a five-day monitoring was about 90 USD. Thus, they concluded that this simple and cheap technique could be used as a routine technique in monitoring free flaps to improve safety of this reconstructive technique.

6.3 AIMS AND OBJECTIVES OF THE STUDY

To study the role of capillary Lactate/Glucose ratio from the flap as a predictor of Flap Survival.

7.0 MATERIAL AND METHODS

7.1 SOURCE OF DATA

A Prospective study will be conducted on all consenting patients who will undergo pedicled flap or free flap surgery in the Department of Plastic Surgery, Dayanand Medical College and Hospital, Ludhiana from August 2021 to December 2022.

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA:

- All the consenting patients of all ages and gender undergoing any kind of flap surgery having accessible skin paddle.

EXCLUSION CRITERIA:

- Patients who do not give consent.
- Flaps that do not bleed on a pin prick.
- Flaps with inaccessible skin paddle.
- All diabetic patients.

The demographic data of the patients including age, gender, diagnosis, site of reconstruction, type of flap as well as need of any intervention or re-operation and final outcome of the flap will be recorded. For any kind of Flap Surgery, the required surgical procedure will be performed. At the completion of surgery, the measurements will be done for capillary glucose and capillary lactate levels from the distal 1/3rd of the flap using 2 devices: one for capillary glucose (Freestyle Optium Neo H) and other for capillary lactate (Lactate Scout 4). The values will be recorded in mmol/l and then the capillary lactate to glucose ratio (L/G) will be calculated.

Measurements will be done at 0,1,3,6,12,24 and 48hrs after surgery, where 0 is immediately post operatively.

Along with L/G ratio measurements clinical evaluation of the flap will also be done at same 0,1,3,6,12,24 and 48 hrs which will include skin paddle colour, capillary refill, temperature and bleeding on pin prick.

Measurements will also be done whenever flap will show any signs of compromised vascularity in between these intervals.

Two groups will be made:

Group 1: Flaps with no post-operative complications

Group 2: Flap with post-operative complications (showing signs of necrosis and , or need of any surgical intervention)

Decision to intervene surgically will be based exclusively on Clinical assessment.

Duration of study: August 2021 to December 2022.

OUTCOME

The L/G ratio of 2 groups will be compared and data will be utilized to calculate the cut off value of L/G ratio at which flaps begin to show signs of non-viability.

Final outcome of the flap and any intervention required will also be noted.

STATISTICAL ANALYSIS: Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test will be performed and exact test was used when the expected frequency is less than 5. Receiver operator characteristics (ROC) curve was done, and criterion value was estimated depending on the specificity and sensitivity. Area under curve (AUC) was measured. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

No

7.4 HAS ETHICAL CLEARENCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3 ?

Attached herewith

8.0 LIST OF REFERENCES

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PROFORMA

S. No. MRD. No.

NAME AGE/SEX

FATHER'S/HUSBAND'S NAME

DIAGNOSIS

SITE OF RECONSTRUCTION:

FLAP NAME:

CLINICAL EVALUATION OF FLAP:

DURATION(HRS)	0 *	1	3	6 *	12 *	24	48 *
COLOUR							
CAPILLARY REFILL							
BLEEDING CHARACTERISTICS							
TEMPERATURE							
OUTCOME OF FLAP							

*Flap Pictures (where ever possible) will be taken at these intervals

Signature of Candidate **Signature of Supervisor**

FLAP GLUCOSE, LACTATE AND L/G RATIO MEASUREMENTS:

DURATION(hrs)	LACTATE (mmol/L)	GLUCOSE(mmol/L)	L/G RATIO	OUTCOME OF FLAP
0				
1				
3				
6				
12				
24				
48				

L/G Ratio measurements whenever flap shows any signs of deterioration in between these intervals:

Signature of Candidate **Signature of Supervisor**

INTERVENTION REQUIRED	DURATION AFTER SURGERY (hrs)	SIGNS OF NECROSIS	L/G RATIO

FINAL OUTCOME OF FLAP (Tick Appropriate)

FLAP SURVIVED	VENOUS INSUFFICIENCY	ARTERIAL INSUFFICIENCY	FLAP NECROSIS

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o **"ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"ROLE OF CAPILLARY LACTATE/GLUCOSE RATIO FROM FLAP TO PREDICT FLAP SURVIVAL" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ